RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES

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Irradiation Therapy in Hodgkin's Disease[1]

CHARLES M. NICE, M.D., and K. WILHELM STENSTROM, PH.D.

THE STUDY OF Hodgkin's disease includes many interesting pathologic and clinical ramifications. One of the most consistent observations in this disease has been that, although the lesions appear radiosensitive, the mortality rate is high. Previous reports from this institution (34, 35) have outlined the general therapeutic approach and given the survival figures obtained in the past. It is the present purpose to bring up to date the results of irradiation therapy in Hodgkin's disease at the University of Minnesota Hospitals and to review fundamental concepts on the nature of this disease and its therapeutic management.

HISTORICAL DATA

Historical reviews of Hodgkin's disease can be found in numerous publications and here will be limited largely to references (2, 16, 17, 18, 22, 26, 32, 38, 40, 49–53, 55, 56). A particularly accurate histologic description of the characteristic cells was given by Reed (40), in 1902.

Many suggestions as to the cause of Hodgkin's disease have been made, but none has been substantiated. The absence of any proved infectious agent, the high fatality rate, and the occurrence of cases with apparent primary lesions and later metastases tend to support the neoplastic theory.

CLINICAL PICTURE

The clinical picture in Hodgkin's disease is variable. Enlargement of single or multiple groups of lymph nodes may be the only complaint. Onset may be heralded by weakness, fever, anorexia, nausea and vomiting, weight loss, or pruritus. Cough, dyspnea, cyanosis, or dysphagia may be indicative of mediastinal lymphadenopathy. Pulmonary parenchymal involvement may be accompanied by fever and the lesions may cavitate. Frequent coincident infections include tonsillitis, upper respiratory, otitic, and oral infections. Vertebral or extradural involvement may produce monoplegia or paraplegia. Backache is commonly caused by enlarged retroperitoneal nodes. Localized pain usually precedes actual roentgen demonstration of bone lesions, and bone marrow studies may disclose multiple granulomas. Jaundice may be due to enlarged nodes about the common duct or to actual hepatic involvement. Enlargement of the spleen, invasion of the stomach and kidneys, as well as involvement of other organs, may be accompanied by clinical findings. Specific skin lesions are not infrequently demonstrated, and herpes zoster occurs in some cases.

PATHOLOGY

Pathologically (2), the lymph nodes

[1] From the Department of Radiology, University of Minnesota Medical School, Minneapolis, Minn. Presented at the Thirty-ninth Annual Meeting of the Radiological Society of North America, Chicago, Dec. 13–18, 1953.

grossly are enlarged, pale, and firm, discrete, and of fleshy or fibrous consistency. They are not easily differentiated from other lymph node tumors. Microscopically, there is an increase in the number and size of the reticulum cells, often with the formation of mononuclear or polynucleated giant cells of the Reed-Sternberg type, increase of reticulum fibers with formation of areas of fibrosis, obliteration of sinusoids, a variable number of eosinophils, areas of necrosis, and increase of lymphoid cells. Invasion of the capsule may be noted. Other organs or tissues involved show similar histologic changes.

Microscopic differentiation from other tumors of lymph nodes is difficult. Jackson and Parker (27) would restrict the diagnosis to those cases showing Reed-Sternberg cells. These latter authors describe three histologic patterns which they consider of prognostic importance: *Paragranuloma*, the early variety, with hyperplasia of adult lymphoid cells, is slowly progressive and may lead, after a variable period of time, to Hodgkin's *granuloma*, which presents histologic evidence of eosinophilia, necrosis, and fibrosis, with more rapid progression and more serious prognosis. The third type, Hodgkin's *sarcoma*, is characterized by large tumor cells and Reed-Sternberg cells, with only occasional necrotic areas. This form is rapidly progressive and fatal in a short time. It is most commonly primary in the retroperitoneal lymph nodes and gastrointestinal tract but rather uncommon in the peripheral nodes. Slaughter and Craver (44) dispute the concept of Jackson and Parker, reporting 14 cases of Hodgkin's sarcoma, with 5 patients surviving three years, 3 patients surviving five years, and 1 seven years. Also, the patients in their general series who survived less than six months presented no consistent histologic picture. The concept is further clouded by the fact that biopsy specimens from different areas may show different histologic characteristics. Obviously, if numerous areas are involved, it is not practical to examine each one histologically.

INCIDENCE

Hodgkin's disease shows no racial predilection. In America, Negroes and whites are affected to about the same extent. From 62 to 70 per cent of the patients are males (various series). All ages are affected, the maximum incidence being in the third decade.

The apparent site of onset, in 75 per cent of the cases, is in the peripheral nodes, predominantly the cervical. Other less frequent sites of origin include the mediastinal and abdominal lymph nodes, lung, skin, and other organs. The peripheral nodes are ultimately involved in about 98 per cent of the patients, and endothoracic structures in roughly two-thirds. Abdominal nodes will be involved in over one-half of the cases, and the spleen in about one-third (as high as 70 per cent in some series). Bone and skin are involved with moderate frequency (15 to 20 per cent of cases).

FORMER APPROACH TO THERAPY

In the previous reports from this institution it was emphasized that treatment of patients should be individualized, with certain fundamental principles being kept in mind. It was noted that the most favorable cases are those in which only one chain of nodes is involved, and in these patients, intensive local irradiation was given after biopsy. In general, 1,000 to 2,000 tissue roentgens in fourteen treatment days was advised, with a larger dose where feasible. With massive mediastinal involvement, initial doses of 50 to 75 r in air were recommended to prevent accentuation of symptoms which might follow hyperemia and edema about the bronchial tree. Large masses of long standing were observed to be more radioresistant than smaller masses of more recent origin and, consequently, more intensive therapy was indicated in the former. In all cases, however, a certain minimum dosage was advised even though a rapid response was obtained before the intended amount was administered. Prophylactic irradiation, based on prediction

of future area involvement, was not recommended.

Other approaches to therapy were also discussed. Thus, Slaughter and Craver (46) treated 5 patients having apparent local involvement by local resection followed by irradiation, with survivals of five, six, eight, eleven, and eleven years. Dessauer (10), in 1907, followed by Chaoul and Lange (6) in 1923, utilized spray or total-body irradiation. In the United States this method was employed by Heublein (21), who in 1931 introduced what has been called the "Heublein method" at Memorial Hospital, New York. In collaboration with Craver and Failla, he devised a method of treating patients with prolonged continuous irradiation with hard rays of low intensity (17 r per day, 0.96 r per hour), the average total dose being 100 r. Additional local therapy was given to each group of enlarged nodes. In 1942, Medinger and Craver (33) reported 94 cases in which this method was used. The five-year survival was 24 per cent, an improvement of 6 per cent over the remaining series of patients treated at that clinic.

A modification of the spray method was used at the University of Minnesota Hospitals on a few terminal cases. The irradiation was at a much higher rate per minute, and two or three treatments were given at 140 cm. distance for a total of 30 r in air. Most patients were unimproved, but in 2 cases remarkable improvement was obtained, lasting long enough so that benefit from local therapy was obtained.

Results of irradiation therapy in the first 185 proved cases treated in this institution were reported as showing a five-year survival of 21 per cent and a ten-year survival of 8 per cent. The average survival following beginning of treatment was 33.1 months.

MORE RECENT CONCEPTS

In the more recent literature there seems to be general acceptance of the neoplastic nature of Hodgkin's disease. Sahyoun and Eisenberg (43) studied 24 cases of Hodgkin's disease and offered a histopathologic and clinical classification:

1. A compactly cellular type which progresses slowly and has a range of maximal life expectancy of forty-eight to one hundred and sixty months.
2. Fibrogranulomatous type with maximal life expectancy of twenty to sixty months.
3. A loosely cellular type which is rapidly progressive and has a maximal life expectancy of twelve to twenty months.

Further histologic detail is described in the original article, but it seems that there is at least a gross similarity to the classification of Jackson and Parker (27), as described above, and that, here again, a relative prognosis is attempted on the basis of the histopathologic picture.

Following the tendency to stage some carcinomas clinically, Peters (39) devised a clinical classification for patients with Hodgkin's disease. She listed the following stages according to the extent of involvement on admission:

I. Involvement of a single lymph node region or a single lesion elsewhere in the body.

II. Involvement of two or more proximal lymph node regions of either the upper or lower trunk.

III. Involvement of two or more lymph node regions of both the upper and lower trunk.

The survival figures showed better correlation with the clinical stage than with any other factor. There was a considerable degree of correlation with the histopathologic picture but this was not as good as that obtained with clinical staging. If the presence or absence of constitutional symptoms is also taken into consideration, an even better correlation is obtained. Peters, therefore, suggested the following clinical classification:

Stage I. Involvement of only one lymph node region or a single lesion elsewhere, with no constitutional symptoms.

TABLE I: SITE OF INITIAL INVOLVEMENT IN HODGKIN'S DISEASE

(224 proved cases)

	Number of Cases	Per Cent
Cervical lymph nodes	134	59.8
Axillary lymph nodes	19	8.5
Supraclavicular lymph nodes	5	2.1
Inguinal lymph nodes	10	4.5
Mediastinal lymph nodes	12	5.3
Mediastinal nodes plus pulmonary infiltration	3	1.3
Abdominal lymph nodes	13	5.8
Skin	11	4.9
Bone (including spine)	6	2.6
Generalized	9	4.0
Breast	1	0.4
Thyroid	1	0.4
Tonsil	1	0.4
	224	100.0

Stage II. Involvement of two or more proximal lymph node regions confined to either upper or lower trunk, with or without constitutional symptoms.

Stage III. Involvement of multiple lymph node regions with or without constitutional symptoms, or acute Hodgkin's disease with no obvious lymphatic involvement.

With this type of staging, she stated that, in her series, she would have had 100 per cent five-year survival in Stage I.

From time to time, attention is called to the relationship between the various tumors and lymph nodes (36). Custer and Bernhard (9) analyzed 1,300 lymphatic tumors, many sampled several times during their progress. These writers state that a rigid classification of lymphatic tumors is artificial and confusing and that their series showed a striking fluidity in histologic pattern, with transitions and combinations that could best be interpreted as indicating a single neoplastic entity having a number of variants.

RECENT ADDITIONS TO THERAPY

Gilman and Philips (15) introduced the nitrogen mustards into the treatment of Hodgkin's disease and allied disorders in 1946. Since that time, numerous reports (3, 37, 42, 45, 48, 54) have indicated distinct temporary palliative effects following use of these agents in patients who were resistant to irradiation, in patients with generalized disease, and as an adjunct to irradiation or other forms of therapy. Gellhorn and Collins (13) studied two series of patients: in one radiotherapy alone was used (65 patients) and in the other radiotherapy and nitrogen mustards were administered in alternating courses (67 patients). No significant difference in the four-year survival rate was found in the two groups. However, the amount of radiation required was less, asymptomatic periods were longer, and the economic burden lighter for patients receiving combined therapy.

In 1950, triethylene melamine (TEM) was given to mice with leukemia and various tumors (5, 31). The first group of patients to be treated by this agent was reported by Karnofsky *et al.* (28). Palliative results were comparable to those obtained by use of nitrogen mustards. However, TEM may be given by mouth, causes less nausea and vomiting, and obviates the difficulties of venous thrombosis encountered in nitrogen mustard therapy. Kravitz *et al.* (29) reported successful palliation in 32 of 36 patients with Hodgkin's disease following TEM therapy.

Radioactive phosphorus (24), arsenic (4), gold (44), and various other agents have failed to show comparable results.

PRESENT CONCEPTS AT THE UNIVERSITY OF MINNESOTA HOSPITALS

The histologic sections of the present study and previous studies of lymphoid tumors have been reviewed, chiefly by Dr. Robert Hebbel of the Department of Pathology. In his opinion (19), it is difficult to separate the lesions of Hodgkin's disease into subgroups, certainly to the extent of prognostic implication.

In the clinical appraisal of the patients, a thorough history and physical examination are followed by biopsy of tumor tissue and, in some cases, by study of the bone marrow. The latter may be advisable in all patients. Table I lists the sites of onset in various parts of the body in 224 patients, and Table II, the total sites of involve-

ment. It is noteworthy that the disease apparently starts in the peripheral lymph nodes in 70.4 per cent of patients and that sooner or later involvement of one or more groups of superficial nodes occurs in almost all patients. The mediastinal and abdominal lymph nodes, lungs, and spleen represent the most frequent internal sites of involvement. It should be emphasized that some patients are still living and some died elsewhere, so that a comparable series of postmortem observations would give a much higher incidence in the internal organs.

TABLE II: SITES OF INVOLVEMENT IN HODGKIN'S DISEASE

(224 proved cases)

	Cases	Per Cent
Peripheral lymph nodes		
Cervical	187	83.5
Axillary	144	64.3
Supraclavicular	39	17.4
Inguinal	101	44.6
Thoracic		
Mediastinal lymph nodes	134	60.0
Parenchymal involvement	65	22.0
Pleural effusion	24	10.7
Pericardial effusion	3	1.3
Heart	1	0.5
Abdominal lymph nodes	111	49.5
Spleen	66	29.2
Skin	35	15.6
Bone	37	16.5
Liver	27	12.0
Muscle	8	3.6
Spine	11	4.9
Kidneys	7	3.1
Face	7	3.1
Breast	6	2.7
Adrenals	6	2.7
Stomach	5	2.2
Thyroid	3	1.3
Nervous system	4	1.8
Parotid gland	2	0.9
Pancreas	2	0.9
Omentum	1	0.5
Eustachian tube	1	0.5
Skull	1	0.5
Scalp	1	0.5
Bone marrow	1	0.5
Peritoneum	1	0.5
Gallbladder	1	0.5
Tonsil	1	0.5
Paranasal sinuses	1	0.5

PROPHYLACTIC AND SEGMENTAL IRRADIATION

In 1939, Gilbert (14) reported a method of irradiating cervical, axillary, mediastinal, abdominal, and inguinal lymph nodes, as well as the spleen, in patients with Hodgkin's disease. He stated that he noted a qualitative and quantitative improvement in his series of patients after beginning the use of segmental therapy.

Peters (39) found that prophylactic irradiation to uninvolved areas gave no improvement in five-year survival figures in Stage I disease, but that 13 per cent and 17 per cent improvement occurred in Stages II and III, respectively. Further, in her series, with few exceptions, the first extension occurred in one of the most proximal lymph nodal regions. She believes, therefore, that prophylactic irradiation is indicated, especially to neighboring lymphatic areas.

Hynes and Frelick (25) recently reported on the use of light segmental therapy (up to 1,000 r) and moderate segmental therapy (1,000 to 1,200 r) to all the major lymph node areas, and sometimes up to 1,800 r to the grossly diseased nodes. Their series was small, but they believe a definite trend to prolonged survival is seen, especially with moderate segmental therapy.

Our practice has been to treat an entire chain of nodes intensively when the area is first irradiated. Since we have not been able to predict the location of recurrent disease, we prefer to await evidence of recurrence, then treat intensively.

RESULTS OF THERAPY

In irradiating patients with Hodgkin's disease, as well as with other malignant conditions, it is essential to attempt to utilize an optimum dosage within certain time limits. The treatment should be individualized. In general, when the disease is localized to one or a few regions, an attempt is made to deliver a minimum of 2,000 tissue roentgens to the tumor in fourteen days. In the cervical region, this can be accomplished by giving 900 r in air to each of three fields. Factors are: 220 kv.p., 0.5 mm. Cu plus 1.0 mm. Al filtration, 1.35 mm. Cu h.v.l., 60 cm. distance. Added filtration and increased distance to achieve better depth dose distribution are used for more deep-seated

TABLE III: SURVIVAL IN HODGKIN'S DISEASE AFTER FIRST TREATMENT

(224 proved cases)

Year	No. of Cases	Years of Survival																			Living
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	22	
1926	2	2	2	1	1	1	1	1	1	1	1	1	1	1	1	1	0	0	0	0	0
1927	3	2	2	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1928	5	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1929	8	4	3	3	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
1930	7	4	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0		0
1931	16	12	7	6	5	3	3	3	3	3	2	1	1	1	1*	0	0	0	0		0
1932	18	13	11	6	6	4	3	3	2	2	1	1	1	1*	0	0	0	0	0		0
1933	15	8	7	7	7	7	5	3	2	2	2	1	1	1	1	1	1	1	1		1
1934	14	11	7	5*	1	1	1	1	1	1	1	1	1	1	1	1	1	1			1
1935	15	9	9	7	6	5	3	3	3	2	2*	0	0	0	0	0	0				0
1936	5	2	2	2	2	2	1	1	1	1*	0	0	0	0	0	0					0
1937	15	9	7	6	6	4	4	4	4†	2	2	2	2	2	1						1
1938	12	10	8	6	4	1	1	1*	0	0	0	0	0	0							0
1939	13	4	3	1	1	0	0	0	0	0	0	0	0								0
1940	14	8	7	7	7	5*	4	4	4	4	4	1									1
1941	10	6	5	5	5	5	5	5	5	3	3										3
1942	18	10	8	7*	3	3	2	2*	1	1*											0
1943	5	5	5	4	3	3	3	3	3												3
1944	4	4	4	3	3	3	3	2													2
1945	6	4	2	2	2	2	0														0
1946	3	3	2	2	2	2															2
1947	9	3	3	1	1																1
1948	7	5	4	2																	2
Cases	224	224	224	224	217	208	205	199	195	177	167	153	140	128	113	108	94	79	61	45	18
Survival		140	110	86	69	52	40	37	31	23	19	10	9	8	6	4	3	3	2	1	
Per Cent		65	49	38	32	25	19	19	16	13	11	7	6	6	5	4	3	4	3	2	

* Contact lost with one case.
† Contact lost with two cases.

lesions. We are now using 250 kv.p., 3.0 mm. Cu h.v.l., and 70 cm. distance. Large masses of long standing may require heavier dosage, but even in the smaller masses, a minimum dose of 2,000 tissue roentgens is desirable. Complete chains of nodes are included in the fields, *e.g.*, the submaxillary, cervical, and supraclavicular chains are irradiated if any node in these areas is involved. So that adequate dosage may be tolerated in areas where disease appears, so-called prophylactic irradiation to other nodal areas is not given.

In patients with widespread disease, less intense or palliative therapy is indicated, largely to ameliorate symptoms. When there is massive involvement of the mediastinal nodes, small doses of 50 to 75 r in air are used initially to obviate possible edematous compression of the tracheobronchial tree. A total tumor dose of 2,000 tissue roentgens is still given within a period of three weeks. Total body or spray irradiation is occasionally given in very small doses to patients with widespread involvement. It is in these patients that nitrogen mustards and TEM may be valuable adjuncts.

Special attention should be paid to spinal cord compression caused by Hodgkin's disease. Laminectomy should be performed without delay if symptoms of paresis develop, and the involved area of the cord should then be treated immediately with x-rays. The remarkable palliation which may be obtained from such procedures is exemplified in a report by Smith and Stenstrom (47) which refers to 11 of the patients included in the present series.

In Table III, the survival data for 224 proved cases of Hodgkin's disease are given. In each instance, the microscopic diagnosis has been re-evaluated for this study. For 208 patients followed five years or longer, a survival of 52 patients, or 25 per cent, is noted. For completeness, a separate survival table (Table IV) is presented for patients in whom the clinical diagnosis seemed quite likely to be Hodgkin's disease. In most of these, a histologic diagnosis had been made but the slides were not available for re-evaluation for this study. The results do not differ remarkably.

To evaluate results of therapy, comparison is made to untreated cases, though, unfortunately, few figures are available. Ewing (11) gave the average survival of untreated cases as about eighteen months. Fifty-two cases of untreated Hodgkin's disease collected by Craft (7) from the autopsy records at the University of Minnesota Hospitals showed a five-year survival rate of 6 per cent from time of onset, with no ten-year survivals.

Results obtained mainly by x-ray therapy at some other medical centers help to show what can be accomplished. Krumbhaar (30) reported a five-year survival of 15 per cent at the University of Pennsylvania. Slaughter and Craver (46), mentioned above, in a series of 265 patients at Memorial Hospital, reported a five-year survival of 17.7 per cent, with an average survival of 33.8 months after treatment. This series included the 94 patients treated with the Heublein method, in which group there was a 24 per cent five-year survival. In Peters' (39) series of 113 patients, the five-year survival was 51 per cent, by far the highest reported to this date. Almost all series show an improvement in treated as compared to untreated cases.

The striking results obtained by Peters bear consideration. As she states, comparison of survival rates from various institutions may vary with the material. In recording survivals according to extent of involvement on admission, she notes an 88 per cent five-year survival in 35 Stage I cases, 72 per cent in 32 Stage II cases, and 9 per cent in 46 Stage III cases. For the patients followed ten years or longer, she reports a 79 per cent ten-year survival in 19 Stage I cases and 21 per cent in 19 Stage II cases. None of the 16 Stage III patients lived as long as ten years. Peters believes that the high survival rate for the entire group was due to

TABLE IV: SURVIVAL IN HODGKIN'S DISEASE AFTER FIRST TREATMENT

(78 clinical cases)

Year	No. of Cases	Years of Survival																				Living
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	24	
1926	2	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
1927	5	3	3	3	3	3	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2*	1	1
1928	4	2	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		0
1929	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		0
1930	7	5	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		0
1931	4	2*	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0		0
1932	6	5	4	3	3	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0			0
1933	3	1	1	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0				0
1934	2	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0					0
1935	3	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0						0
1936	2	2	1	1	1	0	0	0	0	0	0	0	0	0	0	0							0
1937	5	3	3	3	1	1	1	1	1	1	1	1	1	1	1								1
1938	4	1	1	1	1	1	1	1	1	1	1	1	1	1									1
1939	0	0	0	0	0	0	0	0	0	0	0	0	0										0
1940	1	1	1	1	1	1	1	1	1	1	1	1											1
1941	5	3	3	3	3*	2	2	1	1	1	1												1
1942	4	3	2	1*	0	0	0	0	0	0													0
1943	1	1	1	1	1	1	1	1															1
1944	5	5	5	5	2	2	2	2															2
1945	5	3	3	3*	2	2	2																2
1946	2	2	2	1	0	0																	0
1947	4	4	3	2	2																		2
1948	3	1*	0	0																			0
Cases	78	78	78	78	75	71	69	64	59	58	54	49	48	48	44	39	37	34	32	29	23	7	12
Survival		50	40	33	22	16	14	9	7	6	6	5	4	4	3	2	2	2	2	2	2	1	
Per Cent		64	51	42	29	23	21	14	12	10	11	10	8	8	7	5	5	6	6	7	9	14	

* Contact lost with one case.

the remarkably small proportion of Stage III cases.

The patients studied here have accordingly been staged, with the results tabulated in Table V. The staging had to be done by reviewing records and may not be as accurate as if it had been done as each patient was first seen.

From Table V, we note, as did Peters in her series, that the five-year survival rates in Stages I and II differ relatively little, while the ten-year survival rate in Stage II is definitely lower than in Stage I. Although Peters had a 9 per cent five-year survival rate in Stage III, she recorded no ten-year survivals. We note a 10 per cent five-year survival and a 2 per cent (3 of 143 patients) ten-year survival in Stage III.

TABLE V: SURVIVAL ACCORDING TO CLINICAL STAGE

	Five-year Survival		Ten-year Survival	
Stage	No. of Cases	Five-year Survivals	No. of Cases	Ten-year Survivals
I	20	17 (85%)	13	10 (77%)
II	20	18 (90%)	17	6 (35%)
III	168	17 (10%)	143	3 (2%)

In Table VI, average and median survival data for separate stages and the total group are given. Here again is noted a relatively small difference between Stages I and II, with definite decrease, both in survival after treatment and total survival, in the Stage III group. The relatively slight difference in duration of disease before treatment between Stages I and III would seem to indicate a biologic difference in tumor-host relationship, *i.e.*, the tumor is less invasive and spreads less rapidly in Stage I. This should, in turn, make the patients in Stage I more amenable to intensive therapy.

In this study we were particularly interested in the survival rate, and the figures indicate that the life span of the patients is prolonged to a considerable extent by means of reasonable irradiation therapy. For most of the patients, however, treatment is given mainly for palliation, as a means of relieving symptoms and severe malaise for a relatively short period of time. Such benefits cannot

TABLE VI: AVERAGE AND MEDIAN SURVIVAL DATA

	Stage I Years	Stage II Years	Stage III Years	Total Years
Average duration before treatment	1.7	0.6	1.3	1.4
Median duration before treatment	0.6	0.5	0.7	0.7
Average survival after first treatment	9.9	8.6	2.2	3.5
Median survival after first treatment	9.9	8.1	1.0	1.6
Average total survival	11.6	9.2	3.5	4.9
Median total survival	11.8	9.5	2.3	3.3

be expressed in figures but are of great importance. Practically all of the patients improved to a greater or lesser extent. Severe radiation reactions may result if treatments are carried out in routine manner, in order to apply a predetermined dose. It is of special importance to modify the treatment to suit the individual. By watching the daily progress of the patient, and by deferring irradiation when indicated, it has been possible usually to avoid untoward reactions.

DISCUSSION

The evidence presented would seem to corroborate the clinical opinions so well established by Peters (39), *viz.*, that clinical staging is the most accurate method by which we can predict survival in patients with Hodgkin's disease, and that comparison of large series from different medical centers has more meaning if grouping into clinical stages is observed. It has been our experience that patients with malignant lymphoid tumors, regardless of histopathologic diagnosis or grading, may present with localized or disseminated disease. Holmes and Schulz (23) reviewed 500 lymphoma records and selected for study 15 cases with survival, apparently free of disease for five years or longer, in which the lesion at the time of treatment was localized and was treated solely by irradiation, biopsy excepted. When the histologic sections were reviewed, examples of all types of lymphoid tumors were found. Only one was a follicular lymphoma (giant follicle tumor, Brill-Symmers disease), and there were no examples of so-called Hodgkin's para-

granuloma. This latter fact is mentioned because proponents of histopathologic prognosis consider these two tumors the least malignant of the group. Additional evidence of unifocal origin of lymphoid tumors is cited by Hellwig (20), who reports approximately 10 per cent of 135 lymphoma necropsies revealing only localized disease, and by Gall (12), who found 29 of 33 lymphomas of the gastrointestinal tract free of metastases.

Gall (12) and Hellwig (20) state a preference for surgery over irradiation in localized lymphoid tumors. Very few of our patients have been subjected to surgery, but we have noticed a tendency to recurrence in the region operated upon, which leads us to believe that, if surgery is undertaken as the primary procedure, it should be followed immediately by intensive irradiation. This view is shared by Craver (8).

Baker and Mann (1) reported 2 cases of localized Hodgkin's disease treated by surgical excision followed by irradiation. These patients had lived ten and twelve years, respectively, at the time of the report. Rhoads (41) prefers the combination of surgical excision followed by immediate irradiation, in localized lymphosarcoma, a closely allied disease. His opinion is shared by many other cancer experts.

In most of the literature reviewed, the opinion seems to be that primary surgical excision alone is not the procedure of choice in localized lymphoid tumors. Whether a combination of surgery and irradiation is superior to irradiation alone is still a debatable point in lesions of the peripheral lymph node areas. Surgical excision of primary foci in the gastrointestinal tract and lungs seems quite logical, since the definite diagnosis is made quite often during exploratory operation. Laminectomy seems mandatory in the cases in which compression of the spinal cord is present.

Though the duration from first symptoms to treatment was short in Stage III, indicating a more acute type of disease, it seems that great alertness among physicians may bring these patients to treatment before they have reached this stage. The importance of early diagnosis and treatment is clearly brought out by the statistics.

SUMMARY AND CONCLUSIONS

A series of 208 patients with proved Hodgkin's disease showed a five-year survival rate of 25 per cent; for 167 patients followed ten years or longer, the ten-year survival rate is 11 per cent.

Clinical staging is the most accurate aid in prognosis and is necessary in comparing series from various medical centers. For 20 patients in Stage I, the five-year survival rate is 85 per cent; for 13 of these, followed ten years or longer, the ten-year survival is 77 per cent.

In Stage II, there is a 90 per cent survival for 20 patients followed five years, and a 35 per cent survival for 17 patients followed ten years. Thus, the difference between Stages I and II is shown in the ten-year period.

In the much larger group, Stage III, representing those patients with disseminated disease, the five- and ten-year survival rates are 10 and 2 per cent, respectively.

In Stage I, the treatment of choice is either intensive irradiation or, possibly, surgical excision followed immediately by intensive irradiation. In Stage II, intensive irradiation is the procedure of choice. In Stage III, palliative irradiation to reduce tumor masses or relieve symptoms is indicated. It is in the latter group that the nitrogen mustards and triethylene melamine may serve as useful adjuncts.

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SUMARIO

La Irradiación Terapéutica en la Enfermedad de Hodgkin

Después de repasar algunos de los conceptos fundamentales acerca de la naturaleza de la enfermedad de Hodgkin, preséntanse los resultados observados con la irradiación terapéutica en los Hospitales de la Universidad de Minnesota.

La naturaleza neoplásica de la enfermedad de Hodgkin parece estar hoy día generalmente aceptada. Aunque existe considerable correlación entre el cuadro histopatológico y los coeficientes de sobrevivencia, la clasificación clínica por períodos ofrece el mejor método para predecir las sobrevivencias y comparar los resultados de varias clínicas.

En la serie de 208 casos de los Hospitales de la Universidad de Minnesota, la tasa de sobrevivencias de cinco años fué de 25 por ciento. Para 167 enfermos mantenidos en observación diez años o más, la tasa de sobrevivencias de diez años representó 11 por ciento. Analizando esos casos conforme a los períodos clínicos, los AA. obtienen las siguientes cifras:

Período I: Para 20 enfermos, 85 por ciento de sobrevivencias de cinco años; para 13 enfermos observados durante diez años, 77 por ciento de sobrevivencias de diez años.

Período II: Para 20 enfermos, 90 por ciento de sobrevivencias de cinco años; para 17 enfermos observados durante diez años, 35 por ciento de sobrevivencias de diez años.

Período III: En este grupo, mucho más numeroso, que comprende a los enfermos con la dolencia diseminada, las sobrevivencias de cinco y de diez años llegaron a 10 y a 2 por ciento, respectivamente.

En el Período I, el tratamiento de elección es ya la irradiación intensa o posiblemente la excisión quirúrgica seguida inmediatamente de la irradiación intensa. En el Período II, la irradiación intensa es el procedimiento de elección. En el Período III, está indicada la irradiación paliativa para reducir el tamaño del tumor o para aliviar los síntomas. En este último grupo, las mostazas nitrogenadas y la melanina trietilénica pueden servir de coadyuvantes útiles.

DISCUSSION

Franz Buschke, M.D. (Seattle, Wash.): I quite agree with the authors that in those stages of Hodgkin's disease in which involvement appears to be limited to one or two regional lymphatic areas (the essayists' Stages I and II), treatment to these sites should be sufficiently radical to prevent recurrence in the treated field during the patient's lifetime. I do not believe, however, that this can be consistently accomplished with a dose of 2,000 r. In those cases in which we ourselves observed recurrences in previously treated areas, we found that either the dose delivered to the nodes was below the level of 3,000 r or that the field had not adequately covered the entire regional area. In order consistently to prevent such local recurrences, we feel that a dose of 3,000 or 3,500 r in three weeks, or 4,000 r in four weeks should be considered as minimum.

This policy of more radical treatment of Hodgkin's disease in its earlier stages represents one of the main

reasons why it is essential to retain Hodgkin's disease as a clinical entity separate from other forms of systemic lymphoblastic processes which need a less radical approach, regardless of the biological and histopathological relationship of lymphoblastomas of different types.

This more radical approach also answers the ever recurring question of radical surgery *vs.* irradiation in these early stages of Hodgkin's disease. With such radical radiation therapy, the results are at least as good as those following radical surgery, with much less morbidity. Actually, radiological results will be rather better, because we can more easily cover a larger area than the surgeon can excise. It is perhaps significant that the recommendation of this type of surgery usually comes from institutions in which radiation therapy is not given in such a radical fashion.

The results presented by the authors again indicate that old-fashioned conservative roentgen therapy remains the unchallenged method of choice in the treatment of early stages of Hodgkin's disease, while chemotherapeutic procedures are reserved for palliation in later generalized stages.

This presentation, together with previous contributions of the University of Minnesota group, seems to me particularly valuable because of the intent to re-establish some kind of system in a situation that has become increasingly more chaotic during the last decade. With the advent of chemotherapy, treatment of Hodgkin's disease, as well as that of other lymphoblastic processes, has in many places become over-individualized to the point that it has become haphazard and unsystematic. In spite of the great variability of the disease, which of course calls for considerable individualization and clinical judgment in individual situations, the authors have shown that it is possible to recognize certain guiding principles. I would therefore like to plead that we all follow their suggestions and classify our cases according to such proposed staging. Not only will this give us more security in guiding the treatment in an intelligent fashion; it will also make the evaluation of results more meaningful.

Dr. Nice (*closing*): Thank you, Dr. Buschke, for your discussion. In general, we have found that a dosage of 2,000 tissue roentgens given over a period of fourteen days will control local foci of Hodgkin's disease. In very large tumefactions, somewhat larger doses may be required. It must be emphasized that the total number of treatment days is quite important. In radiotherapy for palliation, smaller doses may be used and the total number of treatment days will be reduced.

One mode of therapy that is especially ill-advised is to give a dosage that causes the tumor to diminish greatly in size and then stop treatment. This may be satisfactory for palliation, and, indeed, that is all it is. For each time the local tumor recurs, the sooner radioresistance will be noted. We believe that the radioresistance is most likely due to growth of the tumor; it may possibly be due in part to changes in the tumor bed. Therefore, in Stages I and II the therapy should be intensive, covering adequate fields. If local foci appear later in other isolated areas, these may also be treated intensively. In Stage III, palliative therapy is indicated, since it would be prohibitive to give intensive dosage to all involved areas.

Radiation Therapy of Pancreatitis

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SINCE ACUTE pancreatitis was first described by Fitz in 1889 (9) the diagnosis and treatment of this disease have presented an ever increasing challenge to internists and surgeons. On the whole, radiologists have not played a significant role in its management. Morton and Widger (14), in 1940, were the first to report the successful use of roentgen rays as a method of treatment. They described 3 cases of the acute edematous type in which 250 to 450 r (in air) were given with beneficial effect. Successful response to x-ray therapy has since been reported by others (4, 6, 8, 10, 11, 13, 16). This presentation of the results obtained in 53 cases affords additional evidence of the value of irradiation in the treatment of pancreatitis.

For many years, the designation "hemorrhagic pancreatitis" was used for acute inflammatory disease of the pancreas. The term was criticized by many authors, in that it failed to indicate the tissue death which is the primary lesion in the severest form of the condition. Since the hemorrhage is secondary to necrosis, the terms "acute pancreatic necrosis" and "hemorrhagic necrosis" are more appropriate. A second form generally recognized is the less severe "acute edematous pancreatitis." "Chronic recurrent interstitial pancreatitis" is the term appropriately applied to those cases characterized by exacerbations and remissions over a long period (1).

The classification of pancreatitis into these three categories assists one in arriving at an understanding of the pathologic process and serves as a guide to the prognosis. Since acute edematous pancreatitis is not accompanied by necrosis, hemorrhage, and shock, its response to x-ray therapy is more favorable. Any one of the three types may exist in varying degrees, from mild to severe.

ETIOLOGY

The etiology of pancreatitis has not been definitely established, though undoubtedly the tendency to hemorrhage and necrosis is related to the action of activated pancreatic enzymes. For convenience, the disease may be classified into two groups, etiologically: (1) pancreatitis of infectious origin, the infection being introduced through the blood stream or the lymphatic system or by direct extension from the biliary tree and surrounding viscera; (2) pancreatitis of chemical origin. This second type may be produced (*a*) by reflux of bile into the pancreatic duct following obstruction of the common duct by stones, a tumor, or spasm or fibrosis of the sphincter of Oddi; (*b*) by reflux of duodenal contents; (*c*) by trauma in which pancreatic enzymes are liberated within the organ itself.

The incidence of infection as a primary etiologic agent is not high, and acute pancreatitis in association with the acute infectious diseases is extremely mild, tending to disappear within a short time.

Pancreatitis of chemical origin is based upon the "common channel" theory, *i.e.*, that the pancreatic and common ducts form a communication which permits reflux of bile into the former. Opie (15) was the first to advance this theory, in 1901, after discovering at autopsy an impacted stone at the ampulla of Vater. He was able to force bile into the pancreatic duct by exerting pressure on the gallbladder. Mehnen (12) established the incidence of a common channel at 61 per cent on the basis

[1] From the Department of Radiology, University of Tennessee School of Medicine and St. Joseph's Hospital, Memphis, Tenn. Presented at the Thirty-ninth Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 13–18, 1953.

of autopsy studies. Ordinarily, bile does not enter the pancreatic duct, since the pressure there is higher than that in the common duct. To obtain reflux into the pancreatic duct, therefore, the sphincter of Oddi must be obstructed.

Opie also found that bile injected into the pancreatic duct could produce pancreatitis. Chisholm and Seibel (4) have produced pancreatitis in dogs by a similar procedure. The bile in the ducts leads to edematous pancreatitis. If the injection is made with sufficient force to cause rupture and extravasation, hemorrhage and necrosis ensue.

Pancreatitis is frequently associated with disease of the biliary tract, the incidence of the association varying from 30 to 80 per cent in series of cases reported by different authors. In many cases the pancreatic lesion arises from obstruction of the ducts incident to gallbladder disease, though in others no duct obstruction or reflux of bile can be demonstrated.

Probably the most common cause of pancreatitis is the one most often overlooked, *i.e.*, operative procedures upon the stomach, spleen, gallbladder, and colon (17). It is well to recognize this fact in order that the diagnosis may be suspected in those patients who, several hours after surgery of the upper abdomen, exhibit symptoms compatible with pancreatitis.

SYMPTOMS

Pain, usually severe and continuous, is invariably reported as a symptom of pancreatitis. In the majority of cases, the pain is epigastric and radiates to the back, loin, or subscapular area. Nausea and vomiting are associated in approximately 65 per cent of the cases. A leukocyte count of 20,000 or more is not uncommon, and the temperature may be as high as 103 degrees. A rapid, feeble pulse, cold and clammy skin, and collapse quickly follow. Not all patients manifest such outstanding symptoms, though all have some pain.

DIAGNOSIS

Increasing reports of acute pancreatitis indicate that the disease is far from rare, as was formerly supposed. Thus, it will be more often recognized if kept in mind in the presence of abdominal pain. The diagnosis can be made with accuracy only by laboratory examinations or abdominal exploration. In view of the surgical mortality of 49 per cent, as compared with a mortality of 5.7 per cent among patients who are treated conservatively, every effort should be made to establish the diagnosis without laparotomy.

The most reliable diagnostic measures are the serum amylase and lipase tests (3). The serum amylase is elevated in 87 per cent of the patients and the lipase in 99 per cent. For the highest degree of diagnostic accuracy, both tests are advisable. Results of both can be reported within two to four hours. Amylase studies especially should be obtained early, since the maximum concentration of amylase in the blood is reached within twelve to twenty-four hours after the onset of the disease; it then drops to normal within two or three days, the damaged pancreas being no longer capable of producing amylase. Since the amylase content is also elevated in acute parotitis, renal failure, and obstruction and perforation of the gastrointestinal tract, care should be exercised in the interpretation of the findings.

Roentgen studies have little diagnostic value other than to establish the absence of disease in other organs. Gas may be apparent in the small bowel, but ileus and peritoneal reactions can be caused also by any number of other abdominal disorders. If exploration is necessary and pancreatitis is found, a cholangiogram may reveal some type of obstruction at the ampulla of Vater which may have predisposed to development of the disease.

TREATMENT

Medical management includes opiates, treatment of shock, and fluids to maintain electrolyte balance. Gastric suction to evacuate the gastric and duodenal secretions and prevent distention is often ad-

vantageous. In the presence of suppuration, surgical drainage is sometimes necessary. If cholecystitis is associated with recurrent pancreatitis, removal of the gallbladder may bring about a cure of the latter. For primary pancreatitis of the chronic recurring interstitial type associated with rather severe pain, Connolly and Richards (5) advocate bilateral resection of the sympathetic ganglia from the 7th thoracic to the 3d lumbar vertebra and bilateral resection of the three splanchnic nerves. They have reported relief of pain in two cases, though the period of follow-up was too short to permit an estimate of the effect of the operation upon the disease. Cattell and Warren (2) recommend pancreatoduodenectomy as the best surgical procedure.

The purpose of this paper is twofold. Its first object is to point out that we, as clinical radiologists, should be aware of pancreatitis and should be able to suggest the diagnosis if some other clear-cut entity cannot be established. It is believed that many undetected cases, particularly the subacute type, would be materially benefited by a well organized therapeutic regime.

In the second place, we wish to show that, regardless of the origin or type of pancreatitis, irradiation has a definite place in its therapeutic management. The primary objective is the suppression of pancreatic secretion, which is largely responsible for autodigestion of the organ. That this can be accomplished with deep x-ray therapy has been demonstrated by Rauch and Stenstrom (16) of the University of Minnesota, in experiments on dogs. These investigators found that pancreatic secretion was suppressed for a period of days following irradiation. We should like to suggest that another effect of irradiation is also responsible for recovery, *i.e.*, the same effect generally obtained in the treatment of inflammatory processes in other parts of the body.

Since 1946, we have treated 53 patients by roentgen irradiation. At least one year has elapsed since treatment in each instance; thus, some impression of the permanence of the benefit or the length of remissions is possible.

Not all the patients were treated at the same institution, though the technic followed was similar. X-rays of 2 mm. Cu h.v.l., generated at 200 kv., were used. The ports were always 15 × 15 cm. and the target-skin distance was 50 cm. Two hundred roentgens, measured in air, were delivered daily for three days, the total dosage being 600 r. In 1 of the 53 patients, four series of therapy were required; three series were necessary on 4, and two in 7.

Twenty-four of the patients were males and 29 were females. Their ages varied from 23 to 73 years, the average being 47 years. Twenty-eight had previously undergone surgery: 10 cholecystectomy, 5 gastric surgery, and 13 exploratory laparotomy.

In the majority of cases, the diagnosis was established by clinical-laboratory examinations, and in the remainder by surgical exploration. Lipase studies were not made until recently, when a simplified test was introduced which permitted a report within two hours. In 35 of the 53 patients, the serum amylase was elevated, the highest value being 778 mg. per cent. The usual readings were between 200 and 500 mg. per cent. In 4 patients the amylase level was normal. In 15 no studies were made.

It should be emphasized that some of our cases were less severe than those reported in other series. It was believed that the milder cases were of the acute edematous type. One patient, however, had hemorrhagic necrosis diagnosed by laparotomy and exploration; recovery followed deep x-ray therapy in addition to supportive treatment. Another patient, with a similar form of the disease, expired after exploration and one x-ray treatment. This was the only death in the group which was attributable directly to pancreatitis. In 2 other cases, metastases in the pancreas precipitated attacks of acute pancreatitis. The first of these patients had undergone

resection of the stomach for a carcinoma two years previously, and the second a mastectomy for carcinoma of the breast five years before. Pain, nausea, and vomiting were the cardinal symptoms, and the serum amylase was elevated. X-ray therapy relieved the pain, and subsequent exploration revealed the underlying cause of the pancreatitis. In 2 cases of abscess of the pancreas, surgical drainage and x-ray therapy brought about recovery.

Gallbladder disease was associated in 26 per cent of the 53 cases, as compared to the usually reported incidence of 60 to 80 per cent. This low incidence is ascribed to the fact that many of our patients had a primary mild edematous pancreatitis, which is not generally included in reports of other writers. That gallbladder disease predisposes to pancreatitis is illustrated by the fact that 5 of the patients with both diseases were permanently relieved of the symptoms of pancreatitis following x-ray therapy and subsequent cholecystectomy.

The results in these 53 cases were regarded as good in 33 (62 per cent), fair in 15 (29 per cent), and poor in 5 (9.0 per cent). They indicate the value of roentgen rays in the treatment of acute pancreatitis. It is believed that x-ray therapy, when used early, will prevent abscess formation, necrosis, fibrosis, and possibly recurrences of the disease. It might also render unnecessary such operative procedures as pancreatoduodenal resection and resection of the sympathetic ganglia and splanchnic nerves.

SUMMARY

(1) The etiology, symptoms, and diagnosis of pancreatitis are briefly discussed.

(2) Regardless of the etiology, deep x-ray therapy is advocated in conjunction with supportive measures for all cases of pancreatitis.

(3) The technic and dosage employed in 53 cases have been described.

(4) The results of the treatment were good in 62 per cent of the 53 cases, fair in 29 per cent, and poor in 9.0 per cent.

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(Para el sumario en español, véase la página siguiente)

SUMARIO

Radioterapia de la Pancreatitis

El diagnóstico de la pancreatitis aguda no puede hacerse con exactitud más que con estudios de laboratorio o con la exploración abdominal. Como la mortalidad quirúrgica es elevada (49 por ciento), debe hacerse todo lo posible para establecer el diagnóstico sin la laparotomía. Las medidas más fidedignas de diagnóstico son las pruebas de la amilasa y la lipasa séricas.

Independientemente del origen o de la forma de la pancreatitis, recomiéndase la roentgenoterapia para todos los casos, unida a medidas fortalecientes. Los AA. han tratado a 53 enfermos con los rayos X y presentan los resultados observados al cabo de un período de un año o más. La dosis total administrada en tres días fué de 600 r (200 kv., capa de hemirreducción, 2 mm. cobre; distancia foco-piel, 50 cm.; tamaño de la puerta, 15 × 15 cm.). Un enfermo recibió cuatro series de tratamiento, 4 enfermos recibieron tres series y 7 dos series.

Consideráronse los resultados como buenos en 33 casos (62 por ciento), medianos en 15 (29 por ciento) y malos en 5 (9 por ciento). Opínase que la roentgenoterapia, usada tempranamente, impedirá la formación de abscesos, la necrosis, fibrosis, y posiblemente la recurrencia de la enfermedad.

DISCUSSION

K. Wilhelm Stenstrom, Ph.D. (Minneapolis, Minn.): The poor prognosis of acute pancreatitis and the lack of adequate therapeutic measures has led to the empirical use of irradiation for this disease. Relatively few publications dealing with the results of such treatment are available, though sporadic reports have occurred since R. M. Culler in 1920 referred to two instances of pancreatic fistulas which were permanently closed following x-ray therapy. The paper presented by Drs. Heacock and Cara is, therefore, of importance, especially as it deals with such a large series as 53 patients.

The authors differentiate between three types: "acute pancreatic necrosis," "hemorrhagic necrosis," and "chronic recurrent interstitial pancreatitis." It seems, however, that they had to be satisfied with a clinical diagnosis of acute edematous pancreatitis for some of the patients in their series. In the majority of the cases the amylase level of the blood was measured and was usually found to have a value between 200 and 500 mg. per cent. It may be questioned that the diagnosis can be established by these values. The amylase level is influenced by a number of factors and is elevated, for example, by such drugs as codeine. It may reach 200 mg. per cent even if no pancreatic disease is present. There seem to have been 14 patients included who had neither established amylase level elevation nor surgical exploration. How was the diagnosis made in this group?

The diagnosis was evidently established by means of surgery in 6 patients, and the recovery of 5 of these suggests the value of x-ray therapy The figures reported by the authors with reference to the entire group are impressive. It is unfortunate that no control group was used, with patients receiving the same care but with irradiation omitted. It may be true that serious consequences are prevented by the use of x-ray therapy in the mild cases with clinically diagnosed pancreatitis, but more definite proof is desirable.

The action of radiation is complex, and the beneficial effect may be due to several factors. One of these factors may be the inhibition of enzyme secretion, which would be of special importance in acute necrotic pancreatitis. I had the opportunity to participate in a study of the effect of irradiation on the pancreatic secretion when Dr. Rauch was experimenting with fistulas in dogs. He used a very careful technic and was able to collect the secretion under controlled conditions. He then determined the total volume secreted and, with standard methods, the amylase, the lipase, and the trypsin activities.

The results can be summarized briefly as follows: After a single dose, estimated as 350 to 550 tissue r to the pancreas, no important change in the total secretion or in the pH was noticed. A temporary suppression of the three enzymes was a consistent finding. The amylase activity decreased 28 to 78 per cent after twelve to thirty-six hours in 3 dogs. By the second to fourth day after treatments, the pre-irradiation values were approached. The lipase activity followed a similar course, with a reduction of 50 and 29 per cent in 2 dogs and 45 per cent after a second treatment in 1 animal. The trypsin activity was decreased 30 and 73 per cent respec-

tively, and this depression lasted for three and five days. It seems that such suppression of enzyme secretion should be of importance; that it probably is of similar magnitude and duration in man and perhaps more pronounced when pancreatitis is present.

A different type of response was encountered in a patient with a persistent pancreatic fistula subsequent to marsupialization of a pancreatic cyst. Three treatments of 150 r each were given in four days and resulted in an immediate increase in the secretory volume, followed by a dramatic drop and satisfactory closure of the fistula.

The authors' method of administering 200 r for three successive days, with a half-value layer of 2 mm. copper and a target-skin distance of 50 cm., ought to reduce the enzyme activity. Perhaps one more treatment would be advisable. The report should stimulate further studies of the effect of roentgen therapy in pancreatitis.

Rib Notching Following Subclavian Artery Obstruction[1]

BERTRAM LEVIN, M.D.,[2] and LEO G. RIGLER, M.D.

ATTENTION WAS first focused on rib notching as a roentgenologic sign in 1928 when Roesler (18) reported its appearance in patients with coarctation of the aorta. He later noted (19) that "the erosion of the ribs is the most unequivocal sign (of coarctation of the aorta), although it is not always present nor is it always very marked." In 1929 Railsback and Dock (17), apparently unaware of Roesler's report, published a case of rib notching associated with aortic coarctation, the first to appear in the English literature. They asserted that costal erosion is "undoubtedly pathognomonic of coarctation of the aorta."

This roentgen finding enjoyed the distinction of being a pathognomonic sign for some years. The first evidence that rib erosion could be secondary to causes other than coarctation appeared in 1933, when Hench and Horton (10) reported notching of the inferior borders of the left 7th and 8th ribs in a thirty-three-year-old female who had a loud to-and-fro murmur over the area. There was no clinical evidence of coarctation. The case was thought to represent one of arteriovenous fistula of the intercostal vessels.

In 1937, Laubry and Heim de Balsac (12) discussed the value of rib notching in the diagnosis of coarctation of the aorta; as to it being a pathognomonic sign, they conclude: "*cette assertion est exagérée.*" They reported rib notching in 5 patients without coarctation, as follows: *Case I:* A 48-year-old female suffering from aortic valvular disease and hypertension. Right 5th rib eroded. *Case II:* A 50-year-old male with aortic valvular disease, hypertension, and heart failure. Left 8th rib notched. *Case III:* A 53-year-old male with syphilitic aortitis. Notching of the left 9th and 10th ribs. *Case IV:* Male, 24 years old, with relatively asymptomatic mitral valvular disease. Right 10th rib eroded. *Case V:* Female, age 35 years, with a systolic thrill and a double murmur at the base; blood pressure in the lower extremities higher than in the upper. Right 5th, 6th, 10th, and 11th ribs and the left 10th ribs scalloped by erosion. The authors did not account for the rib changes. McCord and Bavendam (14) suggest that in some of these cases the combination of arteriosclerosis and a wide pulse pressure resulted in pulsations of tortuous intercostal arteries of sufficient force to erode the ribs. They do not attempt any explanation of the changes in the non-hypertensive patients.

Dussaillant, Viviani, and Moya (7) reported the case of a 58-year-old female with generalized arteriosclerosis and pulmonary emphysema. The blood pressure was 150/80 mm. Hg in the left arm and 153/85 in the right. The radial and femoral pulses were strong and equal. Over the left 8th and 9th intercostal spaces in the posterior axillary line there were palpable pulsations and a murmur. The heart size was normal. Roentgenograms showed marked notching of the inferior borders of the left 8th, 9th and 10th ribs. This was thought to be due to intercostal arteriosclerosis. The authors attempt no explanation for the unilaterality or localization of the rib erosions.

In 1948, Holt and Wright (11), in their paper on the radiologic features of neurofibromatosis, reported two cases in which neurofibromas of intercostal nerves produced bilateral symmetrical notching of several ribs. In both cases there were soft-tissue tumors in close proximity to the eroded portions of the ribs. There was

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also incontrovertible clinical evidence of neurofibromatosis.

Piling up further evidence against rib notching as a pathognomonic sign of coarctation, Maier and Stout (13), in their report on arteriovenous aneurysm of the thoracic wall, mentioned a case with arteriovenous fistulas in the chest wall on one side and apparently a pulmonary arteriovenous fistula on the other. The patient had notching of the ribs on the side of the arteriovenous communication in the chest wall.

In 1940, Takaro and Clagett (21) reported the case of a 40-year-old male who presented himself with a pulsatile mass over the posterior left thorax. A bruit was heard over the mass; there was no palpable thrill. A roentgenogram of the chest showed a soft-tissue mass in the left side of the back, with scalloping defects of the inferior margin of the 7th rib, both margins of the 8th rib, and the superior margin of the 9th rib. Surgery revealed a large cavernous hemangioma lying just outside the ribs in the adjacent musculature. Large, tortuous vessels were found lying next to the inferior surface of the 7th rib, the superior and inferior surfaces of the 8th rib, and the superior surface of the 9th rib, all of which were markedly eroded. This was considered to be a congenital arteriovenous fistula large enough to be productive of symptoms and a mass.

In reporting a case of rib notching without coarctation, Batchelder and Williams (3) toll the requiem bell, stating that "it is with considerable reluctance that we herewith record the demise of another 'pathognomonic sign.'" The patient was a 38-year-old male known to have cyanotic heart disease since the age of six years. Final admission to the hospital was because of cyanosis, dyspnea, and orthopnea. There was no cardiac murmur; an apical thrill was present. The blood pressure was 110/90 in both arms and 145/100 in the left leg. Roentgen examination revealed a large heart, engorged pulmonary vessels, and notching of the inferior margin of the left 5th, 6th, and 7th ribs. The patient died soon after admission, and autopsy revealed a classical tetralogy of Fallot. The aorta was of average circumference for its entire length. The orifices of the intercostal arteries on the left side were dilated, measuring 3 to 4 mm. in diameter; those on the right measured 1 to 2 mm. The 5th, 6th, 7th, and 8th left intercostal orifices showed the greatest dilatation. The proximal 5 to 7 cm. of these arteries were irregularly dilated and markedly tortuous, and had caused rib erosions. The right intercostal arteries were straight and of narrow caliber. The left intercostal veins followed the course of the arteries and were slightly dilated as compared to the veins on the right. The internal mammary arteries and veins were straight and of normal caliber. The authors question whether the dilatation and tortuosity of the intercostal arteries may not of themselves constitute a congenital anomaly. Discussing this paper, Gross (9) noted that the dilatation of the intercostal arteries was probably not due to inherent disease in the vessels but to the fact that they carried an increased blood flow as a collateral system. He pointed out that, as people with pulmonary obstruction get older, they begin to build up a collateral circulation. He was, however, unable to account for the unilateral distribution of the dilated vessels.

The intercostal arteries and nerves having been named as causing rib notching, it remained but for the intercostal veins to be implicated. This was done in 1952 by McCord and Bavendam (14). They reported the case of a 59-year-old male with large varicosities of the wall of the chest and abdomen, present since childhood. Roentgenograms revealed marked notching of the right 6th and 8th ribs and minimal notching of all the other ribs from the 3rd to the 9th inclusive. Phlebograms showed complete block of both innominate veins just distal to the expected origin of the superior vena cava, adjacent to several large mediastinal calcifications. Loops of dilated tortuous intercostal veins were seen, the obvious cause of the rib erosion. Autopsy revealed an inflammatory mass

in the anterior superior mediastinum which completely obliterated the superior vena cava. From the veins normally drained by this latter vessel, large anastomotic collateral vessels arose and joined the tributaries of the inferior vena cava. The intercostal veins in the regions of the rib notching were tortuous. The arteries were normal. This report refutes the earlier statement by Fray (8) that dilated thoracic veins secondary to mediastinal tumors do not erode bone.

McCord and Bavendam also report a case of rib notching secondary to dilatation and tortuosity of intercostal arteries, the cause for which was not discovered. The patient was 56 years old and suffering from taboparesis. While he was hospitalized for an acute abdominal crisis, rib notching was noted on a chest roentgenogram. A number of tortuous arteries were palpable in the right lateral chest wall from high in the axilla to the 7th intercostal space. Blood pressure was 126/80 in both arms and 142/100 in both thighs. Heart, lungs, and abdomen were negative. Arteriography revealed normal aortic, innominate, subclavian, axillary, and brachial arteries. The subscapularis was enlarged; the thoracodorsal branch was dilated and tortuous and showed numerous varicosities as it subdivided to anastomose with intercostal arteries. The internal mammary artery anastomosed with the anterior portion of the 5th intercostal artery.

In 1950, Budenz (6) reported a case of tuberous sclerosis exhibiting bony changes. Among other alterations, the long bones showed thickening and irregularity of the cortex. In discussing the chest findings the author states: "There was erosion or 'notching' of the inferior margins of several ribs, similar to that seen in coarctation of the aorta." On the chest film reproduced in the paper, however, the appearance is more that of irregular cortical thickening (as was seen in the long bones) than of true notching of the ribs.

At this time we report a series of 8 cases exhibiting slight to moderate rib erosion due to a cause heretofore unreported. Each patient has had one or more Blalock-Taussig operations attempted for relief of cyanosis and dyspnea due to pulmonic stenosis. In each case the rib notching appeared postoperatively and, whenever this change has appeared, either the surgeon was unable to effect an anastomosis after transecting the subclavian artery on the ipsilateral side or the anastomosis was made but later found to be non-functioning.

CASE REPORTS

CASE 1: S. C., male, born in January 1936, was cyanotic from early infancy. As he grew older, exercise tolerance was markedly diminished; cyanosis became marked on mild exertion, and fingers and toes became clubbed. When the patient was first seen at the University of Minnesota Hospitals at the age of ten years, a loud systolic murmur was heard over the entire precordium, transmitted to the axilla and neck, most intense in the third interspace to the left of the sternum. Blood pressure: right arm 98/70, left arm 94/68, right leg 126/86, left leg 130/90. The clinical and roentgenologic diagnosis was tetralogy of Fallot.

On Oct. 24, 1946, a Blalock operation was attempted on the right side. After the subclavian artery was transected, it was found to be too short for anastomosis with the pulmonary artery; the distance between the bifurcation of the innominate artery and the pulmonary artery was unusually long. The surgeon decided against severing the carotid artery to gain more length. The proximal side of the subclavian artery was ligated and the chest closed.

On May 15, 1947, a Blalock procedure was carried out on the left. No difficulty was encountered in establishing an end-to-side anastomosis between the subclavian and pulmonary arteries. At the completion of the operation, a strong thrill could be felt in the pulmonary artery. Postoperatively the cyanosis was considerably improved, the clubbing diminished, and the boy was on full activity, even participating in school athletics. A machinery murmur was present over the upper left chest posteriorly.

Figure 1A shows the chest roentgenogram taken on March 11, 1948, approximately seventeen months following the right Blalock operation and ten months following the operation on the left. There is no rib notching. Minimal notching of the right 4th and 5th ribs first became evident on March 31, 1949, approximately twenty-two months following the operation. The chest roentgenogram of Sept. 4, 1951 (Fig. 1B) shows moderate notching of the inferior surface of the right 4th and 5th ribs. None is present on the left.

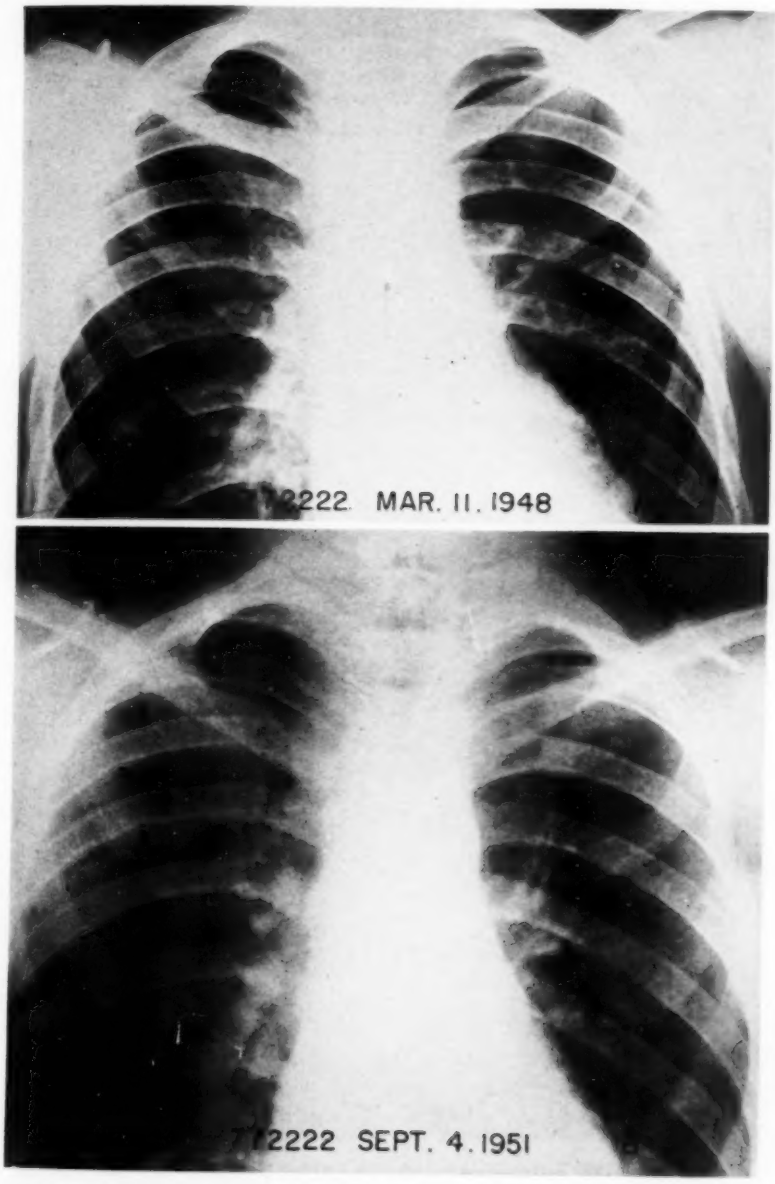

Fig. 1. Case I: Tetralogy of Fallot. A. March 11, 1948, seventeen months following right Blalock-Taussig operation. No rib notching. B. Sept. 4, 1951. Moderate notching of the inferior surfaces of the right 4th and 5th ribs.

CASE II: G. L., male, born in March 1936, was cyanotic from birth and suffered gradually decreasing exercise tolerance. The blood pressure in both arms was 96/68; strong femoral pulsations were present. There was a moderate funnel chest deformity. No cardiac enlargement was evident. A loud systolic murmur was heard in the left 3rd and 4th interspace parasternally, and a soft systolic thrill was felt at the apex at the point of maximal impulse. Roentgen and cardiac catheterization studies established the diagnosis of tetralogy of Fallot.

On June 10, 1949, a Blalock operation was attempted on the right side. The distance between

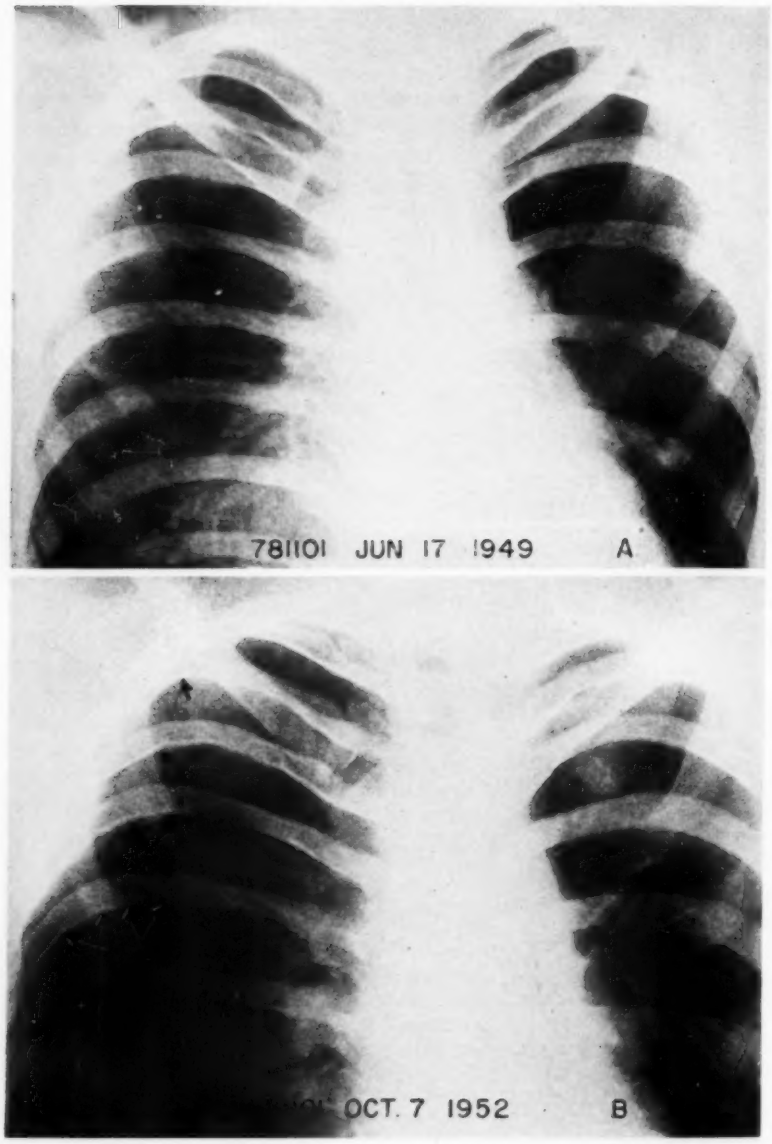

Fig. 2. Case II: Tetralogy of Fallot with pectus excavatum. A. June 17, 1949, one week following right Blalock-Taussig operation. No rib notching. B. Oct. 7, 1952. Erosion of inferior surfaces of right 3rd to 6th ribs.

the subclavian and the pulmonary arteries was considerable. The anastomosis was made, but there was considerable tension on the subclavian artery. When the clamp was removed from this vessel it was found to be crushed at the clamping site. There was no palpable thrill in the pulmonary artery or in the subclavian at the conclusion of the procedure; the attempt to create a shunt had obviously failed.

Following surgery, the patient improved considerably and refused re-operation on the left side. His cyanosis diminished rather markedly, and exercise tolerance increased; he became able to walk a mile or climb three flights of stairs without resting.

Figure 2A is a chest roentgenogram taken on June 17, 1949, one week postoperatively. The displacement of the heart is secondary to a pectus excavatum. No rib erosion is present. On Oct. 7, 1952,

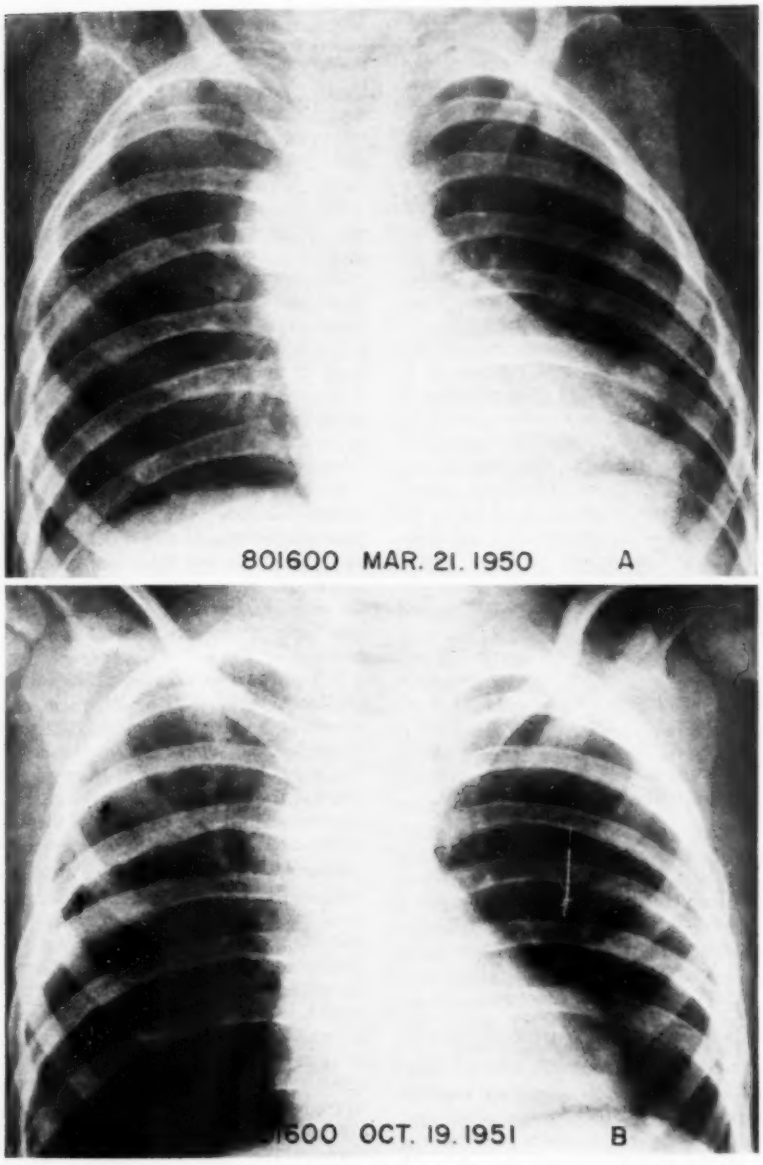

Fig. 3. Case III: Tetralogy of Fallot with right aortic arch. A. March 21, 1950, one month following right Blalock-Taussig procedure. No rib notching. B. Oct. 19, 1951. Erosion of right 4th to 7th ribs.

rib notching first became evident (Fig. 2B). The inferior surfaces of the posterior portions of the 3rd to the 6th ribs on the right (operated side) were eroded. This was three years and four months following the subclavian artery obstruction.

CASE III: S. H., female, born in May 1947, first became cyanotic at about three months of age. Her growth and development were markedly retarded. The heart was moderately enlarged, and a soft, low-pitched systolic murmur was best heard in the second interspace to the right and left of the sternum. The blood pressure was 110/70 in the right arm; femoral pulses were palpable. The roentgen findings indicated a tetralogy of Fallot with a right aortic arch, though the heart was thought to be somewhat larger than is generally seen in this condition. The clinical impression

was tetralogy of Fallot or cor triloculare biatriatum with pulmonic stenosis. Attempts at cardiac catheterization and angiocardiography were unsuccessful.

It was felt that the child was in urgent need of collateral circulation but that she was too small for a Blalock or Potts operation. Pleural scarification was decided upon in the hope that sufficient collaterals could be established between the pleura and the chest wall to carry her along until a definitive operative repair could be done. On March 11, 1949, scarification of the pleura over the left lower lobe and adjacent parietal pleura was done. A section of the parietal pleura was excised.

Following the operation, there was little if any improvement, and the patient was readmitted to the hospital for further study. Angiocardiography was successful and the findings were typical for tetralogy of Fallot. On Feb. 21, 1950, a Blalock procedure was done on the right side and, though there was some kinking of the pulmonary artery, a good thrill was present at the completion of the operation. On the second postoperative day a to-and-fro murmur could no longer be heard and it was assumed that the anastomosis was no longer patent. The patient remained small, retarded, and severely cyanotic.

Figure 3A is a chest roentgenogram of this girl taken on March 21, 1950, one month following the Blalock operation on the right side. There is no evidence of rib notching. The right aortic arch is apparent. Figure 3B, chest film of Oct. 19, 1951, shows moderate erosion of the right 5th and 6th ribs and slight erosion of the right 4th and 7th.

CASE IV: W. P., male, born in October 1949, first became cyanotic at two months. An isolated dextrocardia was present. There were no murmurs or evident cardiac enlargement. Femoral pulsations were normal. The clinical and roentgen diagnosis was isolated dextrocardia with left aortic arch and tetralogy of Fallot.

On April 15, 1950, a Blalock procedure was attempted on the left. The pulmonary artery was very small, and the wall brittle and friable. An end-to-side anastomosis was done. At the upper medial angle of the anastomosis a tear was made in the wall of the pulmonary artery, necessitating deep bites of stitches for repair. A continuous murmur was heard until the eighth postoperative day; it was felt that the anastomosis was then occluded. The operation was followed by some clinical improvement. The intense cyanosis persisted, but exercise tolerance improved and growth and development were accelerated.

Rib notching was first noted on Oct. 20, 1950, six months following the unsuccessful Blalock operation on the left side. The left 4th rib showed minimal erosion.

CASE V: D. M., male, born in December 1941, first became cyanotic and dyspneic at the age of two years. The heart was moderately enlarged and a precordial thrill was present, maximal over the left second interspace. A systolic murmur was loudest over the same area, transmitted to the axilla. Blood pressure: right arm 95/65, left arm 95/65, right leg 110/78, left leg 105/70. The roentgen and clinical diagnosis was tetralogy of Fallot with a right aortic arch.

On June 4, 1947, an end-to-end anastomosis was effected between the right subclavian and pulmonary arteries. The stumps of two branches of the pulmonary artery to the right upper lobe, following their ligation and transection, were used for the anastomosis. At the completion of the operation, a good thrill was felt in the pulmonary artery. Several months later the machinery murmur disappeared. There had been some improvement immediately following surgery, with subsequent regression to the original state of decreased exercise tolerance, polycythemia, and high hemoglobin.

On May 3, 1949, the boy was operated upon again, on the left side. An end-to-side anastomosis was established between the subclavian and pulmonary arteries. Following this there was a good thrill felt in the pulmonary artery. Marked improvement in the exercise tolerance was soon noted, but there was little change in the cyanosis. A machinery murmur on the left side persisted.

Rib erosion first became evident on the chest roentgenogram of Feb. 24, 1949, approximately seventeen months following closure of the first (right) subclavian-pulmonary artery anastomosis. The right 4th and 5th ribs were minimally eroded. Films of the chest taken three years after the second operation showed no notching of the ribs on the side of the successful operation.

CASE VI: J. N., male, born in March 1944, first became dyspneic and cyanotic at the age of six months. There was a loud systolic murmur, best heard over the 3rd left interspace. There was no cardiac enlargement. Blood pressure: right arm 92/60, left arm 90/70, right leg 102/76, left leg 100/78. The clinical diagnosis of tetralogy of Fallot was confirmed by roentgenologic studies. The aortic arch was on the right.

On Jan. 23, 1947, a Blalock operation was performed on the left. The left carotid artery could not be found. The subclavian artery appeared to arise on the right and traverse the width of the mediastinum and then turn abruptly cephalad immediately beneath the pleura. An adequate length of this vessel was obtained by ligating it high in the cupola of the chest, cutting it across, and anastomosing it through an opening made in the left pulmonary artery. Some constriction was noted at the site of anastomosis at the conclusion of the operation, but it was felt that it was adequately patent. Postoperatively, however, no continuous murmur was heard. No improvement was noted following the operation. The exercise tolerance decreased and the cyanosis deepened.

On June 8, 1948, the patient was again operated on. An anastomosis of the right subclavian artery to the side of the pulmonary artery was effected. At the conclusion of the procedure, a distinct palpable thrill was present over the pulmonary artery. The cyanosis and exercise tolerance improved. When the patient was last seen at the University Hospitals in 1951, the continuous murmur was present. He was recalled for a follow-up chest roentgenogram as part of this study, and minimal notching of the right 4th and 5th ribs was noted on the film of Feb. 5, 1953. Physical examination then revealed that the machinery murmur was no longer present. In the interim, there had been no evident clinical regression. The exact time of disappearance of the murmur is unknown. Though the first unsuccessful Blalock operation on the left was done eighteen months before the second, there was still no evidence of rib notching on that side six years later.

CASE VII: B. J. K., female, born in July 1938, was cyanotic from birth. A high-pitched systolic murmur was best heard in the 3rd left interspace. The blood pressure was 95/85 in both arms and 105/80 in both legs. Roentgen studies were in agreement with the clinical impression that this child had a tetralogy of Fallot.

On May 21, 1947, a Blalock-Taussig procedure was carried out on the left side. The left pulmonary artery was quite hypoplastic. A good anastomosis was made, although there was considerable kinking of the arteries. At the completion of the operation a thrill was felt in the pulmonary artery. Postoperatively a massive chylothorax developed on the left, finally disappearing after numerous thoracenteses. From the second postoperative day no continous murmur could be heard, and it was presumed that there had been thrombosis at the anastomosis. The child was discharged from the hospital five weeks after operation. During the interval no to-and-fro murmur was heard by any of the many physicians who listened to the child's chest.

Following surgery, there was improvement in exercise tolerance, but the cyanosis and clubbing remained unchanged. The patient was not seen again until June 26, 1951. Films at that time revealed minimal erosion of the inferior portions of the left 4th and 5th ribs. On the same day, a continuous murmur was distinctly heard, most clearly in the 1st and 2nd left interspaces.

The absence of the continuous murmur in this case, during the postoperative hospitalization period, plus the rib notching noted on the roentgenograms, brings up the possibility of recanalization of the subclavian-pulmonary artery anastomosis. To the authors' knowledge this has never been reported. However, it is well known that, after ligation without transection of patent ductus arteriosus, recanalization occurs in approximately 10 to 20 per cent of cases. It is conceivable, therefore, that the Blalock-Taussig anastomosis in this case did recanalize.

CASE VIII: D. R., male, born in March 1949, was cyanotic and dyspneic from birth. A loud systolic murmur was present, best heard over the pulmonic area. The second pulmonic sound was barely audible. The electrocardiogram showed left axis deviation. Blood pressure was approximately 90/70 in both arms. The clinical and roentgenologic diagnosis was tricuspid stenosis or atresia with pulmonic stenosis.

On Sept. 19, 1950, a Blalock-Taussig procedure was carried out on the right side. An anastomosis was made between a very small pulmonary artery and a friable subclavian artery. At the conclusion of the operation, a minimal thrill was present at the anastomosis site. The day following surgery no to-and-fro murmur could be heard. In spite of closure of the anastomosis, the child improved moderately in exercise tolerance, but there was no evident change in cyanosis.

Films of the chest taken pre- and postoperatively failed to show rib notching until July 9, 1951, approximately ten months following surgery. At this time there was minimal erosion of the inferior portions of the right 5th, 6th, 7th, and 8th ribs. Later films showed the notching to be slowly progressive.

As a control, roentgenograms of approximately 100 patients having had Blalock-Taussig operations were studied. None had rib notching before surgery and, other than one case reported above, none had rib notching following the operation. Case VI was discovered in this manner; in this instance the roentgenographic finding was the first evidence of closure of the subclavian-pulmonary artery anastomosis.

DISCUSSION

It is noteworthy that in these cases the rib erosions occurred only on the side of the subclavian artery obstruction. Closure of a previously patent Blalock-Taussig anastomosis is readily established by the absence of the characteristic continuous murmur; the approximate time of subclavian artery obstruction is thus known in all the cases of this series except Case VI.

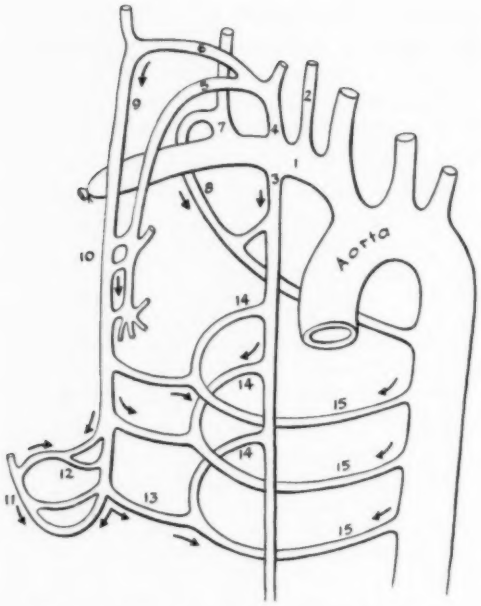

Fig. 4. Schema of collateral circulation following obstruction of the third portion of the right subclavian artery. (Modified from Fig. 525, *Morris' Human Anatomy*, Philadelphia, Blakiston Co., 9th edition, 1933.) 1. Right subclavian artery. 2. Vertebral artery. 3. Internal mammary artery. 4. Thyrocervical trunk. 5. Transverse scapular artery. 6. Transverse cervical artery. 7. Costocervical trunk. 8. Superior intercostal artery. 9. Descending branch of transverse cervical artery. 10. Anastomosis of transverse scapular and descending branch of transverse cervical artery. 11. Subscapular artery. 12. Anastomosis of subscapular artery and descending branch of transverse cervical artery. 13. Anastomosis of descending branch of transverse cervical and posterior intercostal arteries. 14. Anterior intercostal arteries. 15. Posterior intercostal arteries.

We have made no arteriograms, but the mechanism whereby there is increased blood flow through the intercostal arteries in these cases is readily understood when the branching of the subclavian artery is studied (1, 5, 15, 22). When the third portion of the artery is occluded, the blood normally flowing through this vessel is shunted through the thyrocervical and costocervical trunks and through the internal mammary artery. Figure 4 illustrates the anastomotic network. The path of blood flow is much the same as that taken in aortic isthmus narrowing; the changes are, of course, on the side of the subclavian artery obstruction. The internal mammary artery generally arises from the under surface of the subclavian artery at about the same level as the thyrocervical trunk. This artery descends anteriorly and communicates directly with the first five or six anterior intercostal arteries. It terminates by dividing into the superior epigastric and musculophrenic arteries. The latter is directed obliquely downward and laterally and gives off intercostal branches to the 7th, 8th, and 9th intercostal arteries.

The thyrocervical trunk arises from the front of the first portion of the subclavian artery and quickly divides into the inferior thyroid, transverse cervical, and transverse scapular arteries. The transverse scapular artery passes laterally across the root of the neck. It eventually ramifies in the supraspinatus fossa of the scapula, descends to the infraspinatus fossa and terminates by anastomosing with the circumflex scapular artery and the descending branch of the transverse cervical artery.

The transverse cervical artery lies at a higher level than the transverse scapular. It passes transversely and divides into an ascending and a descending branch. The descending branch passes to the medial angle of the scapula and descends along the vertebral border of that bone as far as the inferior angle. It supplies a number of muscles and anastomoses with the subscapular and transverse scapular arteries and with the posterior branches of some of the intercostal arteries.

The costocervical trunk branches from the subclavian artery beyond the origin of the thyrocervical trunk. It soon divides into the deep cervical artery, which extends upwards into the neck, and the superior intercostal artery. The latter vessel is the sole arterial supply for the first intercostal space. It also sends collaterals to the second intercostal space, anastomosing with the first aortic intercostal artery.

Thus the intercostal arteries eventually receive a major share of the shunted blood. Though all the vessels noted above become dilated and perhaps tortuous, the intercostals are the only ones in which the changes are manifest on the roentgeno-

gram made without benefit of intra-arterial contrast medium.

Three of 7 patients previously reported (23) as having non-patent subclavian-pulmonary artery anastomoses showed no rib erosion. The possible reasons are twofold. One, the follow-up period may not have been long enough; two, the major collateral routes may be through channels other than those leading to increased flow through the intercostal arteries. Thus it is that 25 per cent of those with coarctation of the aorta show no rib erosion.

Some of the patients in this series showed improvement even though the attempt to create an artificial ductus failed. It has been known in the past that simple exploratory thoracotomy with the subsequent formation of adhesions and the resulting additional collateral blood supply to the lungs seems to improve the condition of some patients in whom the pulmonary artery is not of suitable size for anastomosis (4). Barrett and Daley (2) recommend removing a portion of parietal pleura to increase the number of collateral channels. This was done in Case III.

It has been the experience of numerous writers that rib notching associated with coarctation of the aorta is seldom seen until after the first decade, although some cases have been reported in which erosion occurred earlier in life. Neuhauser (16) has reported a case with rib notching at nineteen months, and we have personally observed this finding in one case at eight months. In 1949, Shapiro (20) reported clinical studies of a group of patients with coarctation of the aorta. Four patients had well advanced erosion of the ribs at the age of six years. This author comments: "It is obvious that, when extensive erosion can be observed at the age of six, this process must have had its inception some years previously." It is of interest to note that the average interval between the time of subclavian artery obstruction (*i.e.*, the onset of increased blood flow through the intercostal arteries) and the roentgenographically evident rib notching in this series of cases is approximately twenty-three months. In Case IV, only six months elapsed between the subclavian artery obstruction and the appearance of rib erosion.

SUMMARY AND CONCLUSIONS

The major cause for rib notching is undoubtedly coarctation of the aorta. This sign, however, can no longer be considered to be pathognomonic of coarctation. The bony changes may be directly due to local dilatation or tortuosity of intercostal arteries, veins, or nerves. The literature has been reviewed and the many causes for rib notching noted. Some cases with no apparent cause for the rib changes have been reported.

To the growing list of causes for rib notching still another has been added: obstruction of the third portion of the subclavian artery. Eight cases are reported in this series, all secondary to unsuccessful Blalock-Taussig operations. The collateral arterial circulation readily accounts for the rib changes, which are secondary to increased blood flow through the intercostal arteries.

Roentgenographic evidence of ipsilateral rib notching following a Blalock-Taussig procedure should arouse the suspicion that the anastomosis is no longer patent. This finding may be the first evidence of closure of the anastomosis.

ADDENDUM

Since this paper was accepted for publication, a report of two cases of rib notching following unsuccessful Blalock-Taussig operations has appeared (The Development of Rib Notching After Surgical Intervention in Congenital Heart Disease, J. V. Kent, Brit. J. Radiol., **26:** 346–351, July 1953). One patient was a two-and-a-half-year-old male with dextrocardia, pulmonary stenosis, and tricuspid atresia. A subclavian-pulmonary artery anastomosis was made on the left side, resulting in some clinical improvement. Six months later notching was noted in the left 4th, 5th, and 6th ribs, and subsequently the left 8th and 9th ribs also became involved. The author observes that rather more than a year after the operation it was evident that the anastomosis had thrombosed.

The second patient was a nine-year-old male with tetralogy of Fallot, on whom an attempted right

subclavian-pulmonary artery anastomosis was unsuccessful because of too great tension on the subclavian artery. Despite this, the child improved dramatically. Three years later, notching was evident in the right 4th rib and possibly in the 5th as well.

The author attributes the rib notching to intercostal artery dilatation and tortuosity secondary to the subclavian artery obstruction in the same manner as described in the present report.

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SUMARIO

Indentación Costal Consecutiva a Oclusión de la Arteria Subclavia

La principal causa de la indentación costal es sin duda la coartación de la aorta. Sin embargo, no cabe ya considerar ese signo como patognomónico de coartación. Las alteraciones óseas pueden deberse directamente a dilatación o sinuosidad locales de las arterias, venas o nervios intercostales. La literatura pertinente ha sido repasada, anotándose las muchas causas de la indentación costal. Se han comunicado algunos casos sin causa aparente de las alteraciones costales.

A la lista creciente de las causas de la indentación costal, se ha agregado ahora otra: oclusión de la tercera porción de la arteria subclavia. En esta serie, comunícanse 8 casos, todos ellos secundarios a operaciones de Blalock-Taussig, sin éxito. La circulación arterial colateral explica fácilmente las alteraciones costales, que son secundarias al aumento de la corriente de sangre que cruza las arterias intercostales.

Los signos radiográficos de indentación costal ipsilateral después de un procedimiento de Blalock-Taussig deben avivar sospechas de que la anastomosis ha dejado de ser permeable. Dicho hallazgo puede ser el primer signo de la clausura de la anastomosis.

Accessory Roentgen Signs of Coarctation of the Aorta[1]

M. M. FIGLEY, M.D.

NOTCHING OF THE ribs by enlarged intercostal arteries is the best known roentgen sign of coarctation of the aorta. Less well known signs, such as visible aortic deformity and esophageal displacement, assume especial importance when rib notching is lacking. While these have been described before, they are not as widely recognized as their frequency and usefulness warrant. This paper undertakes a survey of these and other roentgen signs in 75 cases of coarctation and presents their anatomic origins as determined by angiocardiography.

HISTORICAL RÉSUMÉ OF ROENTGEN SIGNS OF AORTIC COARCTATION

Roentgen recognition of coarctation was first firmly established in 1928 by Roesler's (1) description of characteristic rib notching, the roentgen sign which has been given his name and upon which radiologists have so long depended. Earlier, Abbott (2) had recognized a small aortic arch, but the prevailing opinion of roentgenology was that stated by King (3) in 1926: "Roentgenograms are disappointing—the chief value of the roentgen ray is the ruling out of aneurysm." Independent of Roesler, Railsback and Dock (4) in 1929 recognized that rib notching was due to enlarged intercostal arteries and indicative of coarctation. They recorded that Meckel had observed notching in a pathologic specimen in 1827, a few years after Paris' initial record of coarctation in 1791 (5). Other signs—wide ascending aorta, small arch, difficulty in outlining the descending aorta, and large pulsations of the left superior mediastinum—were noted by Schatzki and Hallermann (6) in 1930. In the first comprehensive article in English, Fray (7) emphasized the diagnostic value of an indentation or interruption in the contour of the posterior aortic arch in the left oblique view. Ernstene and Robins (8) stated in 1931 that this defect could be seen at the fluoroscopic screen, together with a forcibly pulsating ascending aorta and reduced pulsation of the descending aorta. Wolke (9) completed the initial recording of the basic radiologic signs in 1937, when in an excellent roentgen study he noted indentation in the aortic shadow in both frontal and oblique views, indicating what he considered to be the site of coarctation. He also found esophageal displacement corresponding to the aortic deformity and indicative of the site of stricture. His observations are clearly substantiated by this present study, though they seem to have been generally neglected in the interval.

Other important contributions include Roesler's detailed exposition of the several roentgen signs and in particular their variability (10). His text and that of Kerley (11), though otherwise thorough, fail to describe esophageal displacement clearly. In 1946, Gladnikoff (12) presented his conception of the anatomic origin of aortic indentation. While his conclusions may be questioned, his redirection of attention to the esophagus and to abnormally large mediastinal pulsation is noteworthy. Stauffer and Rigler (13) confirmed his view of the reliability of this latter sign by roentgenkymograms in 1950. Our interest in esophageal displacement is recorded in the 1949 *Year Book of Radiology* (14) in a footnote to a review of an article by Fleischner (15).

The application of contrast examination to coarctation has expanded greatly in the past decade. Early studies were recorded by Blumenthal and Davis (16) and others (17). Numerous articles since

[1] From the Department of Radiology, University of Michigan. Presented as a scientific exhibit at the Thirty-eighth Annual Meeting of the Radiological Society of North America, Cincinnati, Ohio, Dec. 7–12, 1952.

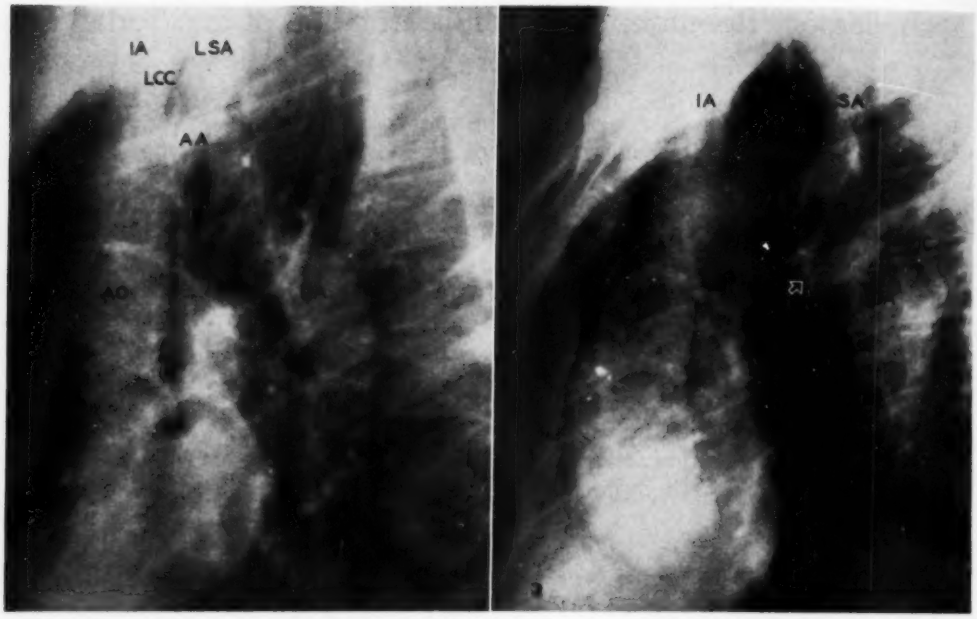

Fig. 1. Coarctation of aorta. Male, 19. Lateral angiocardiogram, showing well developed forward buckling at the coarctation site; large left subclavian artery but no visible intercostals. No rib notching present.

Fig. 2. Coarctation of aorta. Male, 28. Lateral angiocardiogram, showing coarctation at arrow; slight fusiform dilatation of aorta below. Other notable features are dilatation of ascending aorta, long narrow transverse arch, buckling at coarctation site, enlarged left subclavian, internal mammary, and intercostal arteries.

Key to these and following illustrations: AO. Ascending aorta. AA. Aortic arch. DA. Descending aorta. IA. Innominate artery. LCC. Left common carotid artery. LSA. Left subclavian artery. IM. Internal mammary artery. IC. Intercostal artery.

published attest to the commonplace use of angiocardiography, but in none is the pathologic anatomy so beautifully recorded as in the recent superb monograph on thoracic aortography by Jönsson, Brodén, and Karnell (18). The details of their aortograms are excelled only by Bramwell and Jones' (19) postmortem injection study.

Except for the attempts to be reported in this paper, no work has been found applying the information gained from contrast studies to the simpler roentgen methods.

CLINICAL MATERIAL

The original films of 75 patients over the age of two years with coarctation of the aorta have been available for study. In 73 of the cases the diagnosis is clearly established on the basis of clinical findings, with confirmation by angiocardiography, surgical exploration, or autopsy in 49. In 2 older patients the roentgen signs are unequivocally those of coarctation, but limited physical examinations did not suggest or permit this diagnosis clinically. A number of patients under two years of age with proved coarctation have not been considered, since they showed no specific roentgen sign except by angiography. The 75 cases have been reviewed in search of all the roentgen signs of which mention was found in an extensive survey of the literature. Though an objective appraisal was attempted, the incidence of these signs as presented is undoubtedly colored by judgment made in retrospect and a predetermined conviction of the high frequency of several of them.

Angiocardiography and aortography were attempted 48 times in 41 patients. Forty-three satisfactory examinations in 39 patients have been used extensively in anatomic analysis. Most of these studies were made in lateral projection at 36

inches, so that correlation with frontal and oblique projections is necessarily indirect. In most cases, however, the proximity of the coarctation site to the spine makes the vertebral bodies reliable points of reference. Some errors in localization are unavoidable but these indirect correlations are seemingly valid in most instances. The monograph by Jönsson, Brodén and Karnell, in which both frontal and lateral aortograms of several patients with coarctation are shown, substantiates this assumption.

Thirty-two of the patients have had surgical verification of the lesion, and the operative findings of the 28 explored in this hospital have been re-examined. On a number of occasions the author was able to discuss the possible origin of the roentgen findings with the surgeon,[2] at the time the chest was open, with the aortic deformity in direct view. These experiences and a review of operative notes, though valuable in many ways, unfortunately have not materially aided the analysis of the origin of the roentgen observations, because of the difficulties of transferring the visible deformity to the aortic contours of the standard projections.

Several patients died prior to the era of corrective surgery. Only one came to autopsy, and unfortunately no contrast studies were made postmortem, nor was the specimen kept intact.

PATHOLOGY

The gross pathology of the usual case of adult coarctation is generally well known—a short, nearly complete obstruction at or below the ligamentum arteriosum. Many details and variations are thoroughly considered by Abbott (2) and Edwards (20) and need no repetition. Some of these of radiologic importance will be noted later. Other features, seemingly less well known, warrant further emphasis.

In addition to the constriction of the isthmus, the coarcted aorta is often further deformed by a medial and anterior buckling, which may be slight to marked in degree (Fig. 1). The ligamentum arteriosum and the coarctation are closely associated at the apex of this deformity, suggesting that it is due to traction exerted upon the aorta by the ligamentum. This results in two posteriorly and laterally directed convexities with the coarctation site between. Likewise the opposite aortic surfaces are fixed anteromedially in the mediastinum. Such kinking or buckling is well illustrated by Love and Holms (21), Edwards *et al.* (20), and Gross (22), and is emphasized by Gladnikoff in his analysis of the origin of roentgen signs (12, 23).

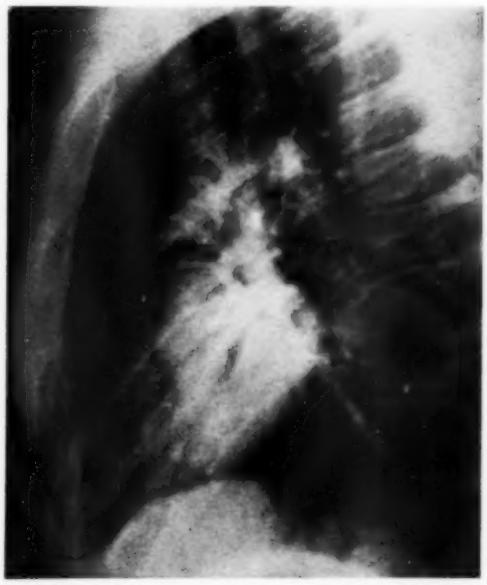

Fig. 3. Coarctation of aorta. Female, 18. Lateral angiocardiogram, showing mild fusiform constriction of aortic isthmus. No visible collateral vessels.

A second feature of radiologic interest is the occurrence of aortic dilatation below the coarctation. This is seldom as extensive as dilatation in the ascending aorta, involving only 3 to 5 cm., but it is equally frequent (Fig. 2). Abbott ascribes it to the entry of blood from dilated intercostals, which are particularly numerous here. Poststenotic aneurysms are not uncommon (4 in this series) and may be an extension of this process or the result of bacterial endarteritis (24).

[2] Dr. Cameron Haight or Dr. Herbert E. Sloan.

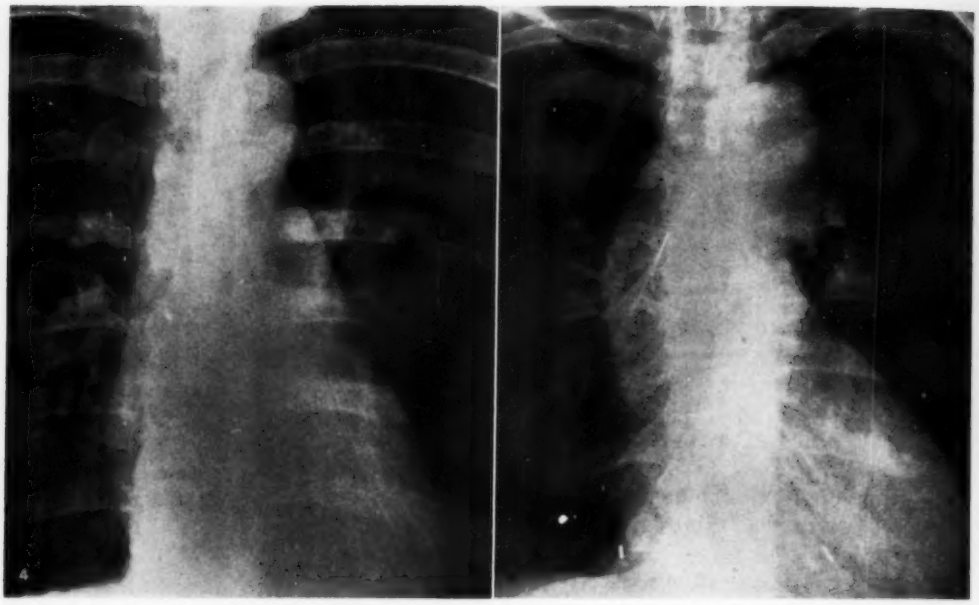

Fig. 4. Normal aorta. Female, 28, with hypertension. Note continuous line defining left margin of arch and descending aorta.

Fig. 5. Slightly dilated and elongated hypertensive aorta. Female, 55. Note continuity of left margin from arch to descending aorta.

Finally, it must be recognized that coarctation is not always severe or accompanied by large collateral vessels. As will be seen later, and as the literature indicates, there are certain cases of mild or moderate coarctation with poorly developed collateral circulation or none at all (Fig. 3). In the two large autopsy series (2, 24), moderate coarctation, defined as a lumen of 0.5 cm. or more in diameter, occurred in 22.5 and 33 per cent of cases. Reifenstein *et al.* (24) noted that in all of their 6 cases with no demonstrable collateral circulation moderate coarctation was present, whereas in none of 21 cases with marked collateral circulation was the coarctation moderate. While it is the severe strictures that are most readily recognized clinically and most suitably treated surgically, certain mild stenoses can be diagnosed both clinically and radiologically.

SIMPLE ROENTGEN METHODS

Aortic Deformity: Careful examination of the non-coarcted aorta in frontal view shows that a continuous line defines the left lateral contour as it is followed from the "knob" down the descending aorta, whether this is medial and straight, as normally (Fig. 4), or lateral and convex, as when the vessel is dilated and elongated (Fig. 5).

By contrast, the coarcted aorta often shows some deviation in this area. The least obvious is an indentation or concavity just below the aortic "knob." This is more abrupt or deeper than normal. Other cases show no indentation, but the descending aorta is slightly convex to the left over a short area below the "knob" (Fig. 6), a contour not normally seen at the relatively early age when most coarctation is discovered. Neither of these features alone is particularly arresting, but they may be combined into a diagnostic contour. In such case, if the descending aorta is followed upward, it will be seen to have a lateral and superior convex margin that terminates in the mediastinum below the aortic "knob" (Fig. 7). The normally continuous lateral margin

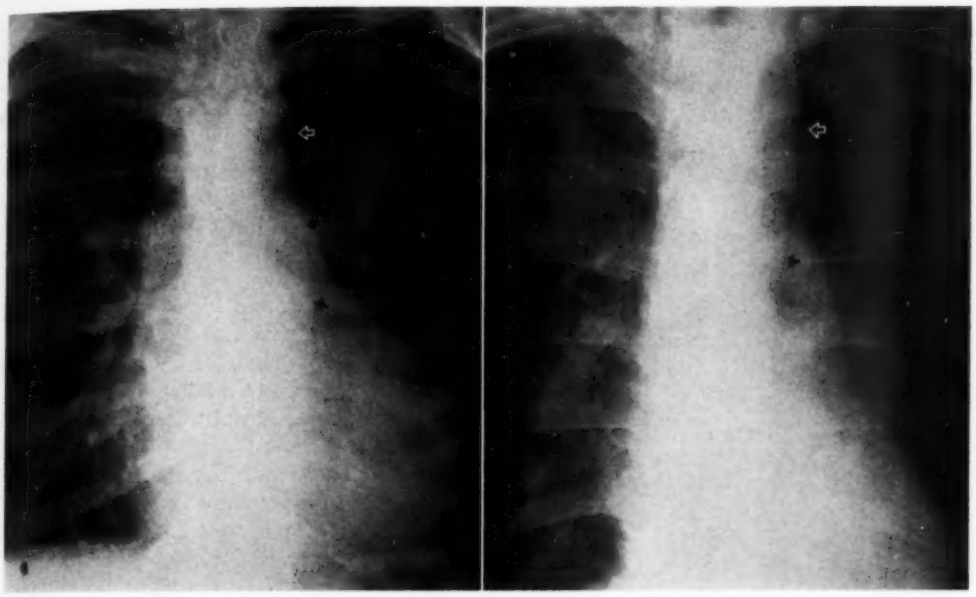

Fig. 6. Coarctation of aorta. Female, 20. Abnormal convexity of descending aorta (lower two arrows) and small aortic "knob" (upper arrow).

Fig. 7. Coarctation of aorta. Male, 20. Note discontinuity of small "knob" (upper arrow) and descending aorta (lower two arrows).

of the "knob" and descending aorta is then distinctly discontinuous. In other instances, the descending aorta is not conspicuous or its contours are normal (Fig. 12). A poststenotic aneurysm is the extreme in development of the abnormal features.

In this study, the upper descending aorta appeared normal in 40 per cent of the cases, abnormal but not diagnostic in 18.5 per cent, diagnostically discontinuous from the arch in 37.5 per cent, and aneurysmal in 4 per cent. A recent article by Bruwer and Pugh (25) describes the same interruption in contour in 34 of 97 cases (34 per cent). It is more consistently seen in older patients, where deformities are better developed.

The left anterior oblique and lateral projections provide a view of the full sweep of the aortic arch and descending aorta. Lack of visibility of the distal arch and descending aorta is commonly cited as a sign of coarctation. Sometimes this is the case, but it is hardly diagnostic, as such invisibility may be noted normally, especially in children. On the other hand, a clear-cut, smoothly convex contour of the upper posterior surface of the aorta, continuing into the descending aorta, is of some value. Such a contour is the rule in hypertension due to causes other than coarctation. Its absence in a patient with hypertension should prompt a search for other, more diagnostic signs of coarctation.

Of more immediate interest is the demonstration of an indentation (Fig. 9) in the posterosuperior aortic contour behind the tracheal air shadow. In this event the indentation marks the coarctation site. Approximately 30 per cent of patients have some visible deformity in the posterior aortic contour.

Mention should be made of the size of the ascending aorta and the aortic "knob." All variations are encountered, large, small, and normal, and none alone is specifically indicative of coarctation. But, much as an inconspicuous distal aorta in hypertension suggests coarctation, so does the association of a large ascending aorta and

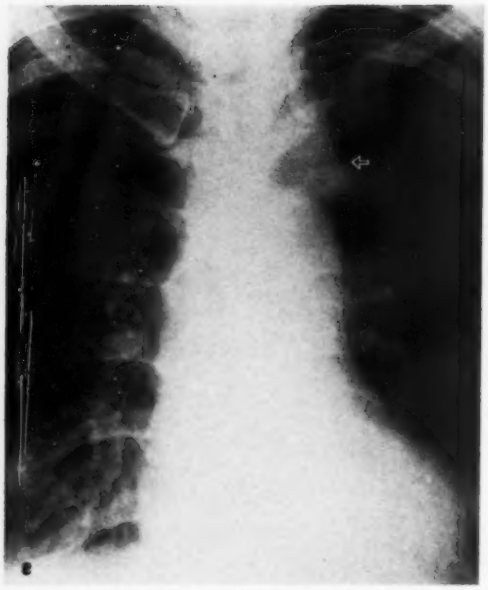

Fig. 8. Coarctation of the aorta. Female, 49. "Double arch contour." True arch above (single arrow), prominent descending aorta simulating second arch (lower three arrows).

inconspicuous "knob." In addition, the "knob" in coarctation sometimes may be higher than expected, especially in a young person, due to some elongation of the ascending portion. In this study, approximatly two-thirds of the patients appeared to have a normally inconspicuous ascending aorta, whereas in one-third this part of the aorta was thought to be enlarged. Perhaps there is some relation of the enlarged ascending aorta to aortic valve insufficiency commonly found in this disease due to an associated bicuspid aortic valve. This relation was not investigated fully. The largest aortas, however, were seen in cases with this valvular lesion.

The vascular structures in the region of the "knob" were arbitrarily judged as more or less prominent than the expected normal: 57 per cent appeared normal (Fig. 12); 25 per cent small (Fig. 6); 18 per cent large (Fig. 8).

Finally, consideration must be given to the well known discrepancy in the forceful pulse of the ascending aorta, "knob," and left mediastinum, and the inconspicuous pulse of the descending aorta. This roentgen equivalent of the cardinal clini-

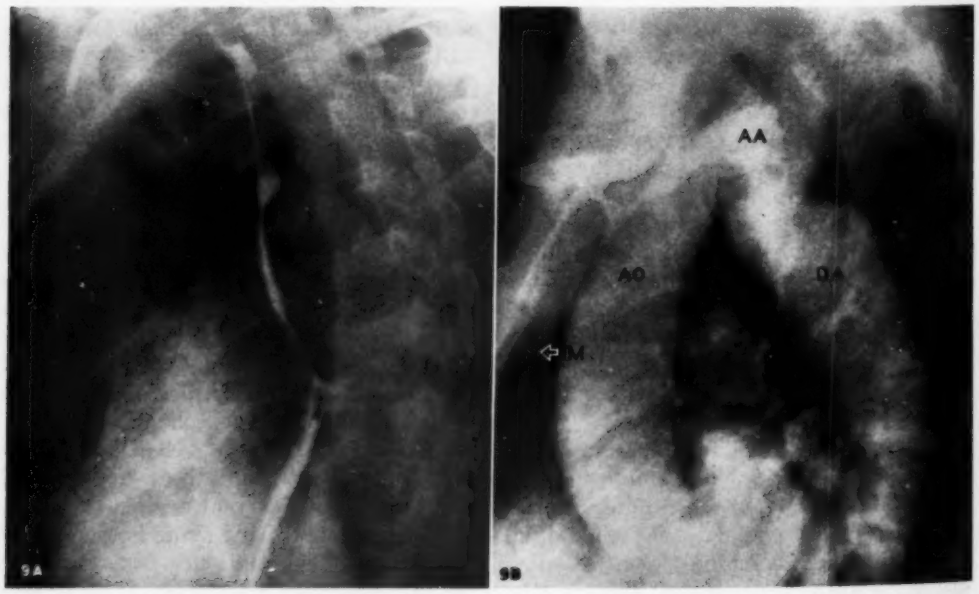

Fig. 9. Coarctation of aorta. Female, 55. Note indentation at arrow in posterior aortic contour (A) corresponding to coarctation site in angiocardiogram (B). Note small internal mammary artery anteriorly. No rib notching.

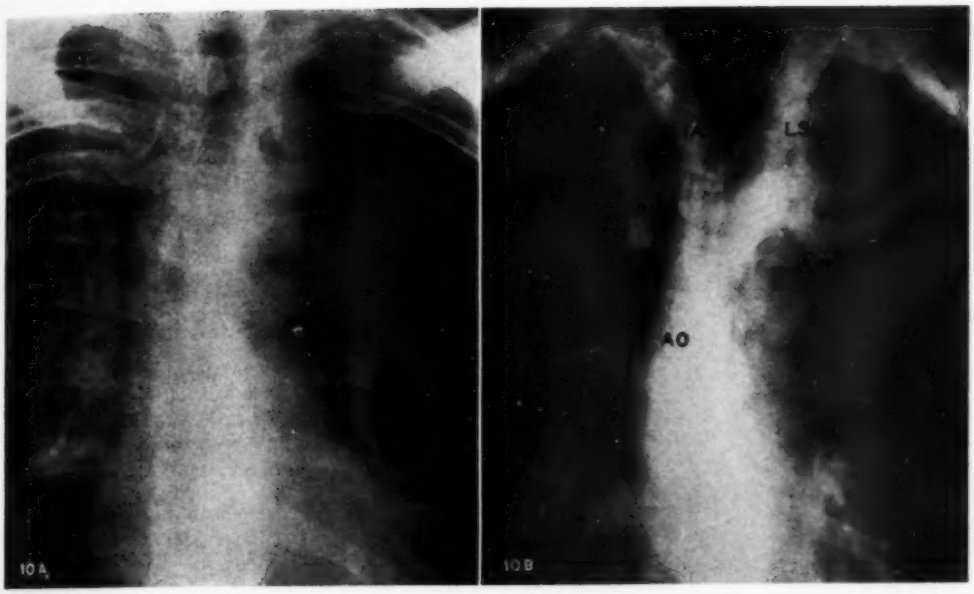

Fig. 10. Coarctation of aorta. Male, 27. Abnormal lateral position of the left superior mediastinal margin (A), due to dilatation of left subclavian artery (B). Prominent "knob" below.

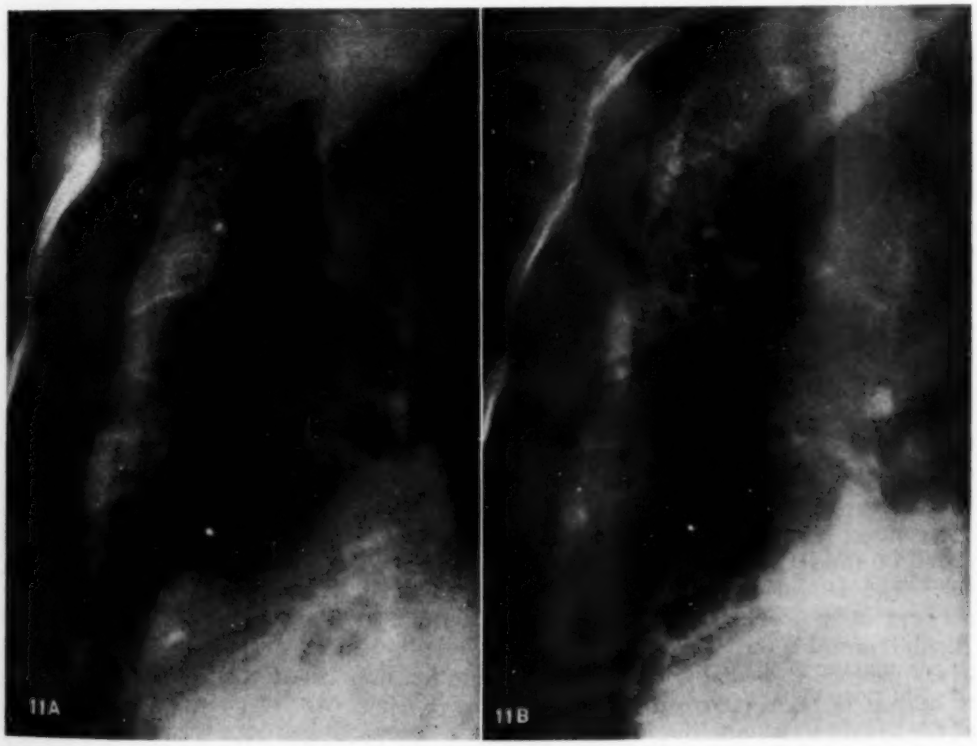

Fig. 11. Coarctation of aorta. Male, 27. Left anterior oblique projection. Note sinuous soft-tissue contours over the sternum (A) corresponding to dilated internal mammary arteries, contrast-filled (B).

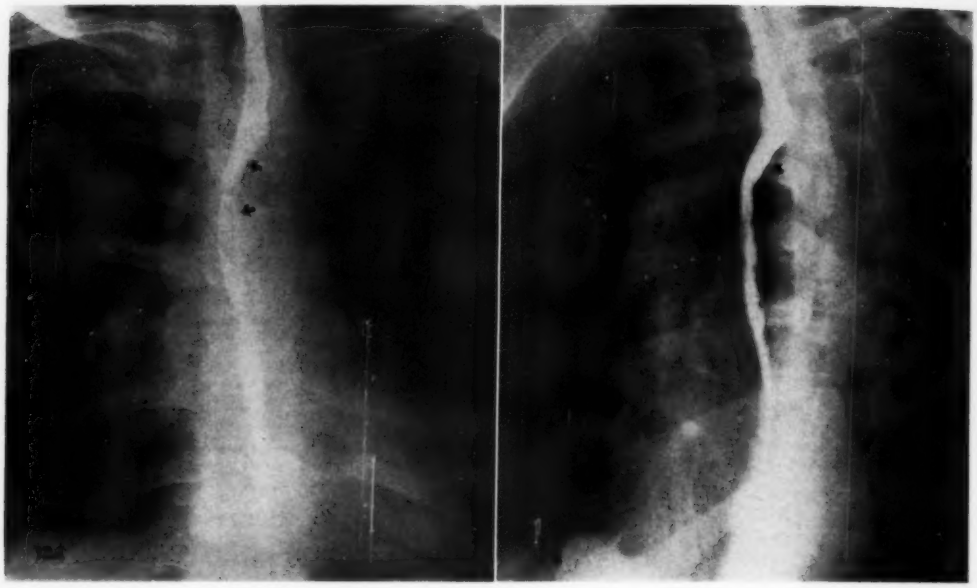

Fig. 12. Coarctation of aorta. Female, 19. Subarch esophageal deviation. Frontal view (A) shows deviation at left of tracheal bifurcation well below expected position of aortic arch. No significant arch impression above. Left oblique view (B) shows same displacement.

cal sign may be seen fluoroscopically or recorded by roentgen or electrokymography (26). The indentation in the frontal and oblique contours proves to be the transition zone, being due to the coarctation. About 50 per cent of this series showed such a pulse deficit. All 4 of the poststenotic aneurysms were non-pulsatile.

Other Abnormal Contours: The normal left margin of the superior mediastinum is slightly concave, and generally there is a sharp demarcation where this contour reaches the summit of the arch. Gladnikoff (12) has emphasized the lateral position of the margin, effacement of the demarcation, and especially convexity of this contour in coarctation, all due to dilatation of the left subclavian artery (Fig. 10). The lateral margin of this artery may actually form the contour of the "knob" and be mistaken for the arch, which lies more medially. It is difficult to be sure of slight degrees of prominence of the left mediastinum, but a convexity is easily recognized as abnormal. Among 71 patients this contour appeared normal in 23, abnormally lateral without demarcation in 39, and distinctly convex, as well as lateral, in 9.

Another interesting feature, and one not previously recorded,[3] is an unusual scalloped contour in the soft tissues immediately behind the sternum, seen in lateral or better in a steep oblique projection (Fig. 11). This proves to be due to enlargement of the internal mammary arteries as they cross beneath the costal cartilages. Once again the change is usually slight and not always recognizable without angiocardiography, but in 21 of 74 patients the retrosternal contour seemed definitely abnormal. Erosion of the lateral scapular margin, attributed to collaterally enlarged scapular arteries, has been noted by MacLaughlin (42). No convincing example was found in our material.

Left ventricular hypertrophy and dilatation may be expected to develop subse-

[3] Since this material was exhibited, Ödman has published an article on this subject, "The Appearance of the Internal Mammary Arteries in Coarctation of the Aorta." Acta radiol. **39**: 47, January 1953.

quent to the hypertension associated with coarctation. Transverse cardiac diameters were judged by tables of prediction based on height and weight. The diameter was normal in 44, slightly increased (+15 per cent) (or the contour suggested hypertrophy) in 16, and enlarged above 15 per cent in 14 patients. Since autopsy studies indicate left ventricular hypertrophy in virtually all cases, these roentgen methods failed to detect it in a significant number.

Esophageal Deviation: It is of interest that deviations in the normal course of the esophagus in coarctation should have been neglected, with some exceptions, for it is a widespread and profitable practice to examine the esophagus in many forms of cardiovascular disease. Wolke (9) apparently was the first to describe the displacements which have been found very helpful in the patients of this study. Subsequently Gladnikoff (12), Fleischner (15), Kerley (11), Gross (22), and others (27) have noted various causes of esophageal deviation.

It is pertinent, before surveying these deviations, to recall Evans' exquisitely detailed account of the normal course of the esophagus, and especially the spatial relation of the esophagus to the aortic arch (28). He emphasized that, particularly in young persons, the esophagus does not lie against the summit of the arch and, therefore, the arch impression cannot be used reliably to determine arch diameter. Very often the normal arch impression in the frontal view begins below the aortic summit, due to the fact that it is related to the right lateral aspect of the distal aorta some 3 cm. beyond the summit. As this more posterior region is the area where coarctation occurs, some abnormality in the intimately related esophagus may be expected.

Although in coarctation the esophageal course is sometimes normal, more often there is deviation to the right and anteriorly well below the arch and the expected level of its impression. This sub-arch deviation, best seen in frontal and left anterior oblique views, may at times be hardly greater than the normal left bronchus impression, whereas in other cases it is particularly conspicuous (Fig. 12). An abnormal course of this sort was considered to be indicated in the frontal view in 50 per cent of cases, and in the left oblique in 80 per cent. Equally frequent (75 per cent) is a small arch impression. This is conspicuous by contrast if the "knob" happens to be particularly prominent and serves to distinguish coarctation from other forms of hypertension where the arch impression is consistently prominent.

Esophageal displacement as described here has been simulated in some normal children and young adults and in a few cases of patent ductus arteriosus. In the former, the relation of the esophagus to the upper descending aorta is responsible; in the latter some enlargement of the aorta at the ductus insertion may be the cause (29). Mediastinal lymphadenopathy

TABLE I: FREQUENCY OF THE VARIOUS ROENTGEN SIGNS OF AORTIC COARCTATION

(Figures in parentheses indicate number of assessable cases)

Rib notching (74)	
Present	77%
Absent	23%
Left mediastinal contour (71)	
Normal	32.5%
Lateral	55.0%
Convex	12.5%
Retrosternal scalloping (74)	
Present	28.5%
Left ventricular size (74)	
Normal	60%
Slightly enlarged or hypertrophied	21%
Moderate or markedly enlarged	19%
Esophageal course	
Subarch deviation	
Frontal (34)	53%
Left oblique (42)	80%
Small arch impression (28)	75%
Aortic contour	
Descending (66)	
Normal	40.0%
Abnormal, but not diagnostic	18.5%
Discontinuous	37.5%
Aneurysmal	4.0%
Deformity in left oblique (52)	30.0%
Ascending (73)	
Normal	67%
Prominent	33%
Aortic "knob" (72)	
Normal	57%
Small	25%
Large	18%
Pulse deficit (36)	83%

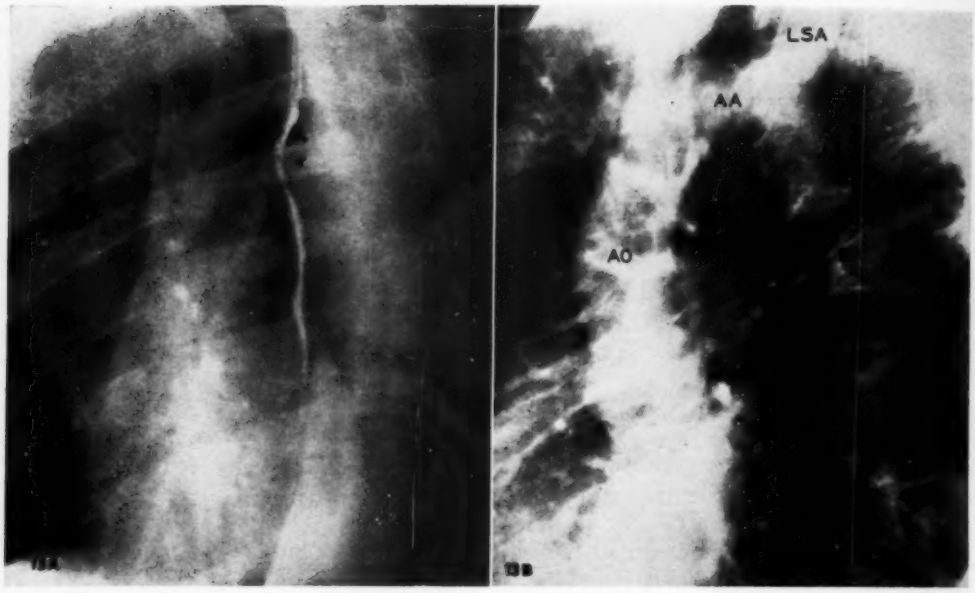

Fig. 13. Coarctation of aorta. Male, 39. Left anterior oblique views. Slight esophageal displacement (A) at level of slight aortic buckling (B). No dilatation of descending aorta.

other aortic disease, etc., may be expected to simulate this finding at times.

Table I summates the frequency of the various signs of coarctation visible by simple roentgen methods.

CORRELATION WITH ANGIOGRAPHIC STUDIES

Forty-three satisfactory angiograms of 39 patients were available for correlation with the aforementioned signs. Each portion of the aorta and the signs relating to it were critically studied. These are considered in order from the heart peripherally.

As might be anticipated, an abnormally convex or lateral ascending aorta proved to be dilated. The angiograms showed the dilatation to be usually slight to moderate, involving the lowermost portion just above the valves (Fig. 2). The anterior and right walls were affected, while the posterior wall usually remained straight or concave. In the 2 most severe cases, aortic valve insufficiency was present clinically, suggesting bicuspid aortic valves. About 25 per cent of normal appearing aortas were actually slightly dilated, making the total incidence of aortic dilatation about 50 per cent, as was found by Abbott. Sometimes the aorta was conspicuously elongated, with a high position of the arch but no dilatation. Dotter and Steinberg's measurements of normal aortas (30) were used for this evaluation.

The angiograms show that a consistent feature of coarctation is the small caliber of the transverse aorta between the left carotid and subclavian arteries (Figs.1 and 2). Normally this portion is nearly the same size as the ascending aorta, whereas in coarctation it is definitely smaller. In all 17 cases with a normal ascendens, the diameter of the transversus was one-half to three-quarters as great. In 13 cases with an enlarged ascendens, the transversus was less than half as large in all but 2. In all severe constrictions, the diameter was about that of the left subclavian artery. Presumably this is due to the fact that in these instances this artery was the only outlet for blood in that part of the arch. It is clear from the angiograms that the small aortic arch im-

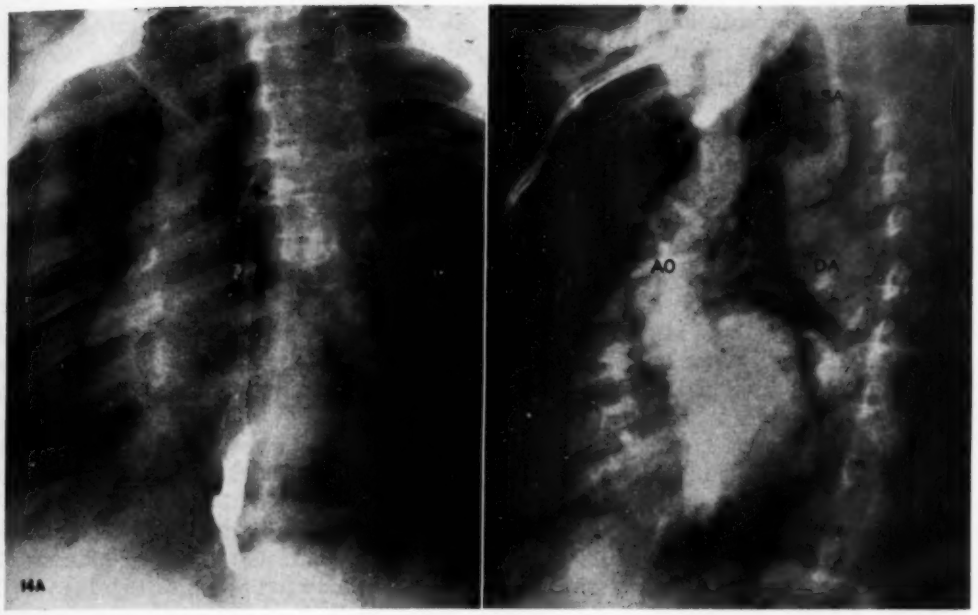

Fig. 14. Coarctation of aorta. Male, 10. Left anterior oblique views. Slight esophageal displacement (A) due to kinking and dilatation of descending aorta (B). Note large internal mammary arteries and very short distal arch above the coarctation (arrow). Slight rib notching.

pression on the esophagus is not a true reflection of the size of the transverse arch, small though it may be. This is because the esophagus lies more posteriorly in relation to the distal portion of the arch. For this reason, the impression cannot be used to define any aortic diameter in coarctation, but its consistently small size is notable.

As we have seen, the structures making up the aortic "knob" are usually normally prominent, but small and even large "knobs" are seen. This study fails to explain these variations, as insufficient frontal angiograms are available. Jönsson's monograph shows the base of the left subclavian artery to be border-forming in some cases. In others the distal arch just above the stricture makes the lateral contour. We have found no correlation between the "knob" size and the size of the left subclavian, the diameter or length of the arch, either in transverse or distal portions, or the degree of kinking. Probably there are multiple factors governing "knob" size, and it seems unlikely that this feature has any consistent anatomic implication.

The distal part of the arch, that segment between the left subclavian artery and the coarctation, is of variable length, shape, and diameter, but of consistent surgical importance. For this reason Jönsson *et al.* (18) classified their cases upon the basis of this variability. Surgically, a long broad lumen (Fig. 1) offers easier resection and the possibility of better clinical result. A short segment may make for some surgical interference by the left subclavian artery (Fig. 14) but, what is more important, if the caliber is small, a limit is placed on the lumen that can be obtained. A particularly narrow distal arch may not allow much clinical benefit unless it can be resected and the base of the subclavian incorporated in the anastomosis. In an occasional case, we have been able to suspect correctly the length of the distal arch, but generally the simple methods fail and angiography of some type is necessary for an accurate appraisal of this important segment.

The origin of indentation in the posterior aortic contour has been controversially presented in the literature. Fray (7), the first to describe this, attributed it to the coarctation site, but more recently Gladnikoff (23), on the basis of surgical observations in 7 cases, held the angle at the origin of the left subclavian and distal arch to be responsible. He argued that the coarctation site was extrapleural with correspondingly little surface deformity. In this respect, our operative notes indicate that the coarctation site is often immediately apparent once the lung is retracted, showing that it has significant surface deformity. Likewise, our contrast studies show that it is the coarctation site which most often causes this indentation (Fig. 9). There are cases, however, in which the angle at the origin of the subclavian seems responsible, and in one case the angle at the origin of a large intercostal was visible. Though this indentation, when visible, usually corresponds to the coarctation and indicates its position in the thoracic cage, it does not define the length of the stricture, a feature also of surgical importance. Elongated strictures may require aortic grafts for successful repair.

Considerable importance has been given earlier to abnormalities in the contour of the upper descending aorta, and especially to the discontinuity of its lateral margin with that of the "knob" above. The angiograms clearly indicate that this discontinuity is a reflection of the coarctation and that the blunt upper end of the descending aorta may be mistaken for the transverse arch, especially if the "knob" is inconspicuous (Fig. 7). Should the "knob" be prominent, a double arch, one above the other, is simulated (Fig. 8). This prominence of the upper descending aorta is the result of slight fusiform dilatation over a 3 to 4-cm. area just below the coarctation, which occurs in the majority of cases. Such dilatation can be suspected if this part of the aorta is lateral in position or convex in contour. In those cases where the descending aorta is not visible, it may still be slightly dilated, but equally often it is of smaller than normal caliber. Medial position may be responsible for this discrepancy. The forward buckling of the aorta previously noted is also an important factor in the double arch phenomenon, for it provides a horizontal surface on the upper end of the descending aorta which may be mistaken for the summit of the true arch.

The cause of what seems to be diagnostically useful displacement of the esophagus is apparently dual in nature. It will be remembered that the esophagus usually lies against the right anterior aspect of the distal arch and upper descending aorta. Thus, either anteromedial buckling or poststenotic dilatation might be expected to displace it. In 31 assessable cases, displacement was found in 25. It seemed to be due to dilatation in 8, to kinking in 11 (Fig. 13), and to both in 6 (Fig. 14). In 6 cases without esophageal displacement, dilatation alone was present in 2, the remaining 4 cases showing neither dilatation nor kinking. While this esophageal displacement is of some value in diagnosis, its dual origin does not allow reliable prediction of the degree of kinking or dilatation, since either one or both may be responsible. In our cases we have not encountered esophageal displacement by enlarged intercostals or other anomalous arteries as reported by others (22).

It might be expected from this discussion that the esophagus would prove troublesome at surgery by its proximity to the aorta. Actually, it is not always isolated, and never in these cases has it been damaged in the dissection of the coarctation site. This disparity of the roentgen and surgical findings would seem to detract from the validity of the foregoing discussion. It is of concern and not understood.

Postoperative studies are unfortunately limited to a few cases. Seldom has the esophageal displacement changed significantly. This might be expected, as the contour and position of the aorta are probably not greatly altered, though it is shorter and probably less kinked. No

helpful postoperative angiograms are available. We have, however, noted restoration of continuity in the left aortic margin where previously it was discontinuous.

DISCUSSION

It is well established that rib erosion is a good sign of coarctation, being both frequent and relatively specific. It occurs in 75 per cent of patients over two years of age (24, 31) (Table I). Though isolated cases of neurofibromatosis (32), anomalous intercostal arteries (33), superior vena caval obstruction (34), aortic valvular disease (35), thoracic wall hemangiomas (36), and obliterative arteritis of aortic branches (37) are reported with rib notching, these instances are sufficiently rare that notching can be considered a reliable sign of aortic coarctation.

In the remaining 25 per cent of cases, the roentgen signs described above may become the only means of radiologic diagnosis. In assessing their value, one may ask their frequency in cases without notching and may inquire as to the clinical significance of absence of the latter feature.

Two groups of patients have been sorted out, 17 who showed no rib notching and 12 with notching so slight that it was or might have been overlooked. In the first group aortic deformity was detected in 11 of 16 cases and esophageal displacement in 8 of 15. Only 5 showed no roentgen sign of any kind. In several cases where the clinical suspicion of coarctation was known, unequivocal roentgen diagnosis based on these signs was eventually proved correct.

In the group of 12 patients with minimal notching, 9 showed aortic deformity and all 7 in whom esophageal study was done showed some displacement. Such evidence adds considerable diagnostic support to notching otherwise equivocal. It is clear from these observations that, if coarctation is suspected, it may be recognized roentgenologically in over half the cases where rib notching is questionable or lacking.

The clinical significance of the absence of rib notching merits discussion. The principal causes reported in the literature are youth of the patient (31), associated patent ductus arteriosus (10, 11), unusual position of the stricture (43), and mild stenosis (24). From the surgical point of view, lack of notching is important, as it suggests collateral circulation too poorly developed to allow the complete occlusion of the aorta necessary for surgical repair. It may also indicate an asymptomatic mild stricture for which correction is not necessary or desirable, at least with the present surgical risk.

Of the 17 patients without notching in this series, 7 were fifteen years of age or younger. Notching takes some time to develop and, though it is occasionally seen in early life, it generally appears at about twelve to fourteen years (Taussig, 38). Collateral circulation may be well developed before notching occurs, as is shown in Figure 14. In this patient and 3 others under fifteen years, in whom notching was either slight or absent, aortic repair was successfully accomplished. Thus, at this age lack of notching does not preclude surgical correction.

In the same group were 3 patients with associated patent ductus, a lesion said to inhibit the development of collaterals. Since all of these were under fifteen years of age, the relation of the open ductus to the lack of notching cannot be established. In all 3 cases good collateral circulation was present at surgery in spite of the patent ductus.

Unusual locations of the coarctation are recorded as being associated with peculiarly distributed rib notching. The notching may be limited to the right side if the stricture lies proximal to or involves the origin of the left subclavian artery. This may be suspected if the blood pressure is lower in the left arm than in the right (39). Our 2 cases, one of which is recorded elsewhere (41), were recognized by this observation. According to Bahnson (43), notching limited to the lower ribs occurs with coarctation in the lower thoracic aorta. We have seen enlarged lower inter-

costal arteries with abdominal aortic occlusion but no associated rib notching.

The principal cause for lack of notching after the age of fifteen seems to be a mild or moderate coarctation. In their report of a large autopsy series, Reifenstein, Levine, and Gross (24) reported: "Rib notching was present in 75 per cent of these 43 cases (cases with chest films), only 10 per cent of which had moderate coarctation; on the other hand, 80 per cent of cases without rib erosion had moderate coarctation." The close relation between the development of collateral circulation and the severity of the coarctation was also brought out in this study.

Available evidence, clinical, surgical, or angiographic, suggests that such a mild stricture was present in 5 of the 10 older patients without rib notching. In none was there any evidence of collateral circulation. One man had a thoracotomy for anticipated aortic repair, but the lack of collateral circulation (shown by preliminary aortography) was considered a serious hazard to aortic occlusion and repair was not attempted. The actual severity of the stricture is known in only 1 of these 5 cases (Fig. 3). Another patient, a woman of fifty-five, proved to have mild coarctation after an asymptomatic "mediastinal mass" (a markedly deformed aorta) was fully investigated (Fig. 9). Similar subclinical cases have been recorded (40).

The remaining 5 patients with no notching all gave angiographic evidence of some collateral circulation, principally enlargement of the left subclavian artery. The stricture was moderately severe in 2 of these. In another (Fig. 1) the stricture was severe (2 × 5 mm. lumen). The collateral circulation, although poorly developed, was adequate for surgical resection.

It is clear from this analysis that mild or moderate coarctation can be recognized clinically and radiologically by simple means. In these cases rib notching is generally absent and collateral circulation may be insufficient for aortic repair. In this same group with no notching, however, a few cases occur which are severe enough to warrant correction and for which there is adequate collateral circulation. The need for differentiation is clear. Angiocardiography has proved reasonably accurate in defining the state of the collateral circulation and in some cases showing the severity of the stricture. In other cases, overlapping shadows prevent accurate delineation of the coarcted lumen. On the other hand, when rib notching is well developed, severe stricture and adequate collateral are assured. In these cases, angiocardiography is principally of value in demonstrating the variable anatomy of the coarcted area.

SUMMARY

A roentgen study of coarctation of the aorta was made to determine the frequency of variously described signs, to clarify their anatomic basis, and to assess the significance of absence of rib notching. Films and records of 75 patients two years or older were reviewed. Angiographic studies in 39 and surgical experience in 28 were correlated with the findings of the simple roentgen examinations.

The most useful accessories to rib notching are visible aortic deformity and esophageal displacement. Varying degrees of characteristic subarch esophageal displacement due to aortic kinking and poststenotic dilatation associated with small arch impression are found in 80 per cent of cases. Rib notching, minimal or equivocal to marked, is seen in 77 per cent. Changes in the upper left mediastinum due to a dilated left subclavian artery may have as high an incidence as 65 per cent but are much less often diagnostic. Over half the cases will show diagnostic pulse deficit if this is searched for carefully. The descending aorta is recognizably abnormal in about 50 per cent of cases and diagnostically discontinuous with the knob along the left margin in 38 per cent. The left ventricle is detectably enlarged in 40 per cent of cases. The ascending aorta is abnormally prominent in 33 per cent but actually dilated in 50 per cent. Thirty

per cent of cases have an indentation in the aorta in the left oblique view, usually at the coarctation site. Abnormal retrosternal soft-tissue contours due to dilated internal mammary arteries may occur in as many as 30 per cent of the patients but only rarely are these marked enough to be diagnostic. The aortic "knob" is large or small in 20 to 25 per cent. Poststenotic aneurysms were found in 4 per cent.

Surgically important variables are the size and shape of the distal aortic arch and the length and contour of the coarctation. Neither of these can be reliably determined by simple roentgen examination. Some form of angiography is necessary for their delineation.

Twenty-three per cent of patients have no rib notching. About two-thirds of these cases can be reliably detected by aortic deformity and esophageal displacement. Under fifteen years of age absence of notching is of no surgical importance, as severe stricture and an adequate collateral circulation are usually present. Over fifteen years, lack of notching may well indicate a mild stricture with minimal collateral circulation, for which surgical resection may be unnecessary or impractical. Yet in this same group with no notching, severe strictures suitable for surgical repair do occur. On occasion, angiography may be of some help in selection of such cases.

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SUMARIO

Signos Roentgenológicos Accesorios de la Coartación de la Aorta

En este repaso de 75 casos de coartación de la aorta prestóse atención especial a los signos roentgenológicos distintos de la indentación costal. Correlacionados los hallazgos arrojados por el simple examen radiológico con los estudios angiográficos en 39 casos y las observaciones quirúrgicas en 38 casos, se llegó a las siguientes conclusiones.

Los accesorios más útiles que apuntan a crenación costal son la deformidad aórtica y el desplazamiento esofágico visibles. En 80 por ciento de los casos, obsérvase en mayor o menor grado típico desplazamiento esofágico debajo del cayado debido a acodadura y dilatación postestenótica de la aorta asociadas con impresión débil del cayado. En 77 por ciento, nótase indentación costal, que varía de mínima o dudosa a notable. Las alteraciones de la porción izquierda del mediastino superior debidas a dilatación de la arteria subclavia izquierda pueden alcanzar una incidencia hasta de 65 por ciento, pero son mucho menos a menudo diacríticas. Más de la mitad de los casos revelarán, si se busca cuidadosamente, un defícit diagnóstico del pulso. La aorta descendente es claramente anómala en 50 por ciento aproximadamente de los casos y está diagnósticamente separada del nudo aórtico a lo largo del borde izquierdo en 38 por ciento. El ventrículo izquierdo está visiblemente hipertrofiado en 40 por ciento de los casos. La aorta ascendente proyecta anormalmente en 33 por ciento, pero está en realidad dilatada en 50 por ciento. Treinta por ciento de los casos muestran indentación de la aorta, que aparece en la vista oblicua izquierda, por lo general en el sitio de la coartación. Hasta en 30 por ciento de los enfermos puede haber contornos anormales de los tejidos blandos retrosternales, debido a dilatación de las arterias mamarias internas, pero esto raramente alcanza intensidad suficiente para que sea diagnóstico. El "nudo" aórtico es mayor o menor de lo normal en 20 a 25 por ciento de los casos. Descubriéronse aneurismas postestenóticos en 4 por ciento.

Factores variables de importancia quirúrgica son el tamaño y la forma de la porción distal del cayado de la aorta y la longitud y el contorno de la coartación. Ninguno de estos puntos puede determinarse con seguridad por medio del simple examen roentgenológico, necesitándose para la demarcación la angiografía en alguna forma.

Veintitrés por ciento de los enfermos no tienen indentación costal. Unas dos terceras partes de estos casos pueden descubrirse seguramente por la deformidad aórtica y el desplazamiento esofágico. En personas de menos de 15 años de edad, la falta de indentación no posee importancia quirúrgica, pues suele haber estrechez intensa y adecuada circulación colateral. A partir de dicha edad, la falta de indentación puede indicar bastante bien una estenosis leve con mínima circulación colateral, para lo cual la resección cruenta tal vez sea innecesaria o irrealizable. Sin embargo, en este mismo grupo (sin indentación) se presentan estenosis graves apropiadas para la reparación quirúrgica. A veces, la angiografía puede ser de alguna ayuda en la selección de esos casos.

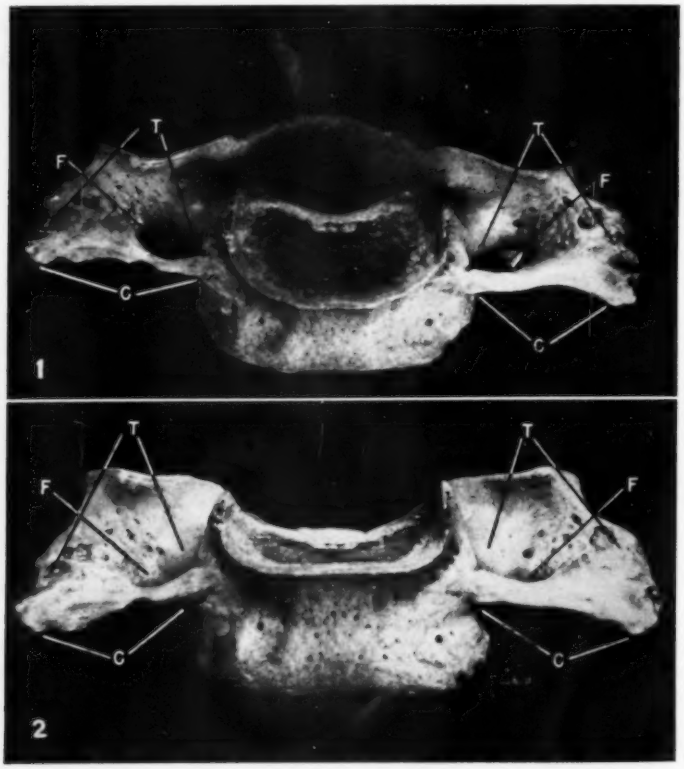
A Source of Potential Error in the Roentgen Diagnosis of Cervical Ribs[1]

D. R. KEATING, M.D., and J. R. AMBERG, M.D.

CERTAIN NORMAL variations in the size and configuration of the costal processes of the seventh cervical vertebra may be deceptive enough to suggest a diagnosis of rudimentary cervical ribs when none is present. This is particularly true during the first ten years of life.

Symptomatic cervical ribs are rare in childhood, but an occasional young patient will be encountered in whom such a diagnosis is entertained on clinical grounds. On these occasions an exact knowledge of the anatomy and development of the seventh cervical vertebra is necessary. Moreover, growth and development of the lateral appendages of this vertebra are in themselves interesting phenomena for observation and study.

The lateral masses of the normal adult seventh cervical vertebra consist of two parts each: anteriorly lie the costal processes, and posteriorly the transverse processes. These lateral appendages of the vertebra are fused at their tips in such



Figs. 1 and 2. Normal seventh cervical vertebra from an adult. Anterosuperior (Fig. 1) and anteroposterior (Fig. 2) views. C. Costal processes. T. Transverse processes. F. Costotransverse foramina.

[1] From the Department of Radiology, Western Reserve University School of Medicine and University Hospitals of Cleveland, Cleveland, Ohio. Accepted for publication in June 1953.

a manner as to form and encircle the costotransverse foramina (Fig. 1). In the adult these foramina lie in the horizontal plane, and as a result of their position they are seldom visible in routine chest roentgenograms (Fig. 2). In childhood, on the contrary, the costotransverse foramina of the seventh cervical vertebra and their surrounding processes are frequently seen in good detail (Figs. 4 and 7). This is explained by the relatively diminutive size of the transverse processes during the childhood period (Fig. 3) and by the fact that the costal and transverse processes are as yet unfused (Fig. 7).

The costal processes of the seventh cervical vertebra develop from ossification centers independent of the transverse processes, and each can often be recognized as a separate unit prior to their mutual fusion. This fusion usually occurs at some time between the fourth and tenth years (Fig. 5).

Close observation of the costal and transverse processes during growth and development reveals variation in their relative lengths. In some children the costal processes are shorter than the transverse processes; in others they are of equal length; in still others the costal processes are the longer. It is in the latter individuals that a false diagnosis of rudimentary cervical ribs may be made (Cases 1 and 2). Examples of such long costal processes are commonly encountered.

In all children there is a differential rate of growth between the costal and transverse processes of the seventh cervical vertebra. At an early age the costal processes may extend well lateral to the transverse processes, but at or near the time of fusion these processes will often have become of equal length, forming normal lateral masses (Cases 1 and 2). This is because during growth and development the transverse process tends to increase slightly in length but considerably in mass, while the costal process always remains of the same relative size and configuration which characterized it at birth. This differential rate of growth makes it hazardous to specify the presence of rudimentary cervical ribs during the first decade of life, when relative length of costal and transverse processes is used as the only criterion. In some individuals the costal processes of the seventh cervical vertebra are small; sometimes they are absent. The transverse processes are of much less variable size.

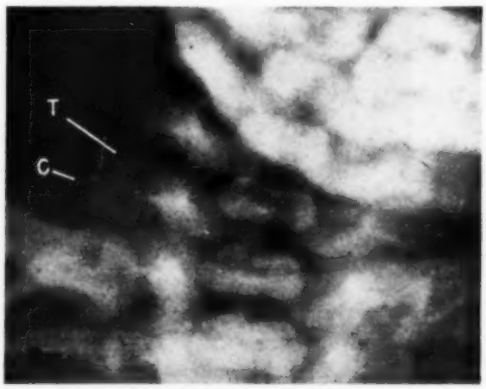
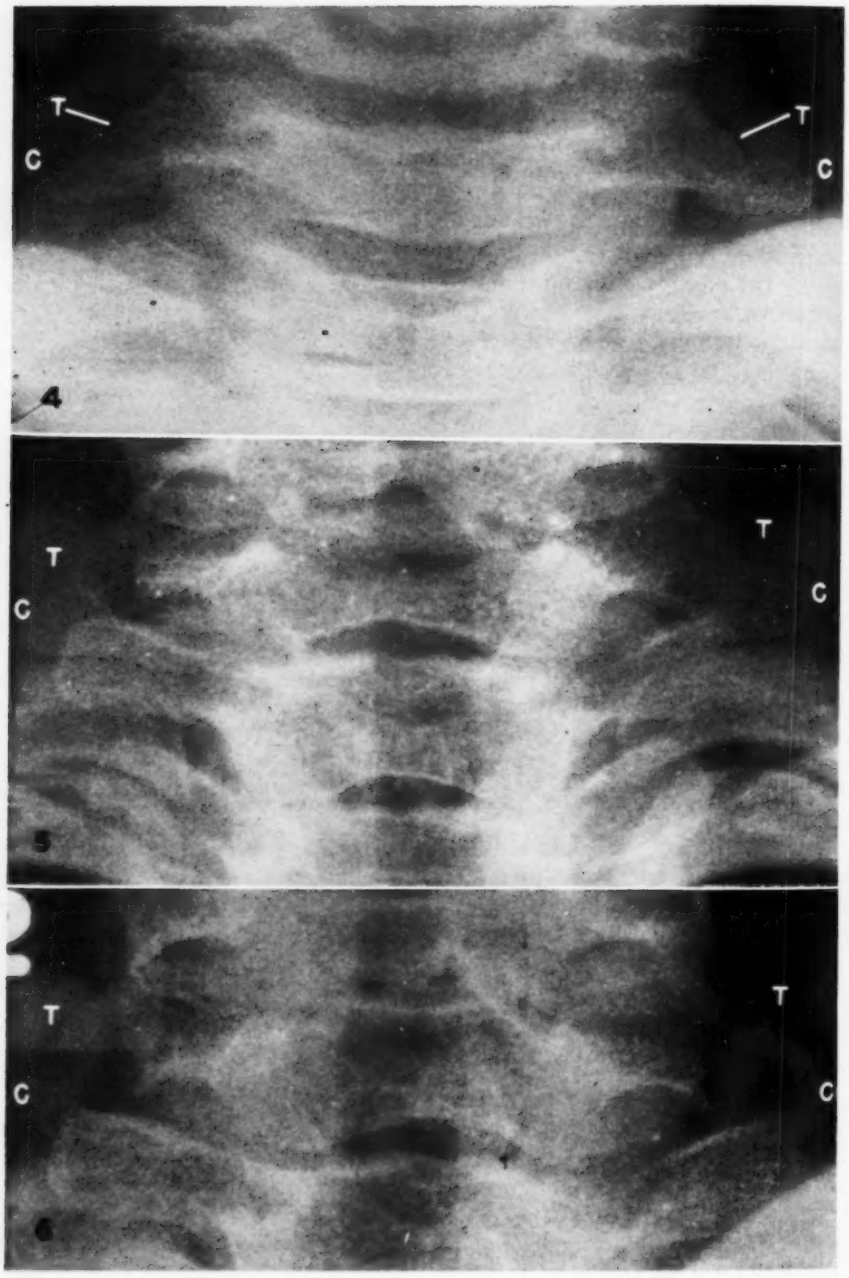


Fig. 3. Normal newborn. The costal process of C-7 is well ossified (C). It extends lateral to the transverse process (T) and is independent of it.

It will be recalled that cervical costal processes, cervical ribs, and normal thoracic ribs are identical in embryogenesis. Distinction is made between them on the basis of length, configuration, and relation to individual vertebrae. When fusion of costal and transverse processes does not exist in the adult, the diagnosis of rudimentary cervical ribs may be made with ease. In children, relatively great length of the costal processes is often a poor criterion of cervical ribs. Satisfactory evidence of cervical ribs must include the anterior and downward curvature exhibited by all true ribs (Cases 4 and 5).

DISCUSSION

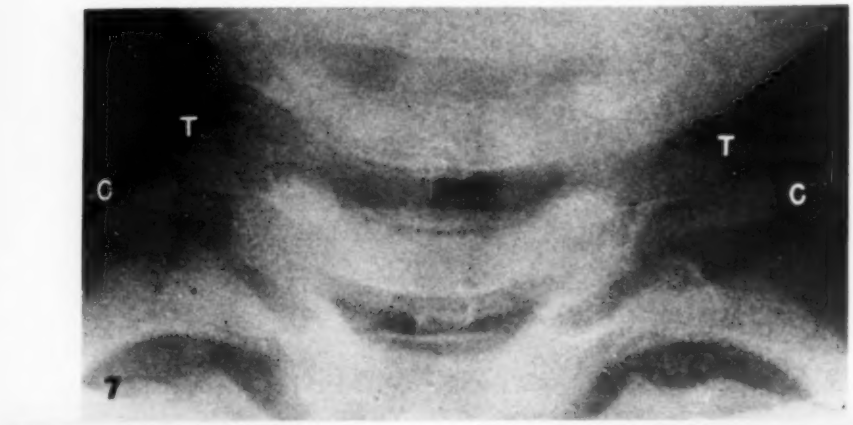
In view of the similar embryogenesis of cervical ribs and normal thoracic ribs, one would expect cervical ribs, when present, to be well developed at the time of birth, just as are thoracic ribs. We believe that complete ossification of cervical ribs, like thoracic ribs, occurs during prenatal life. Our own material does not

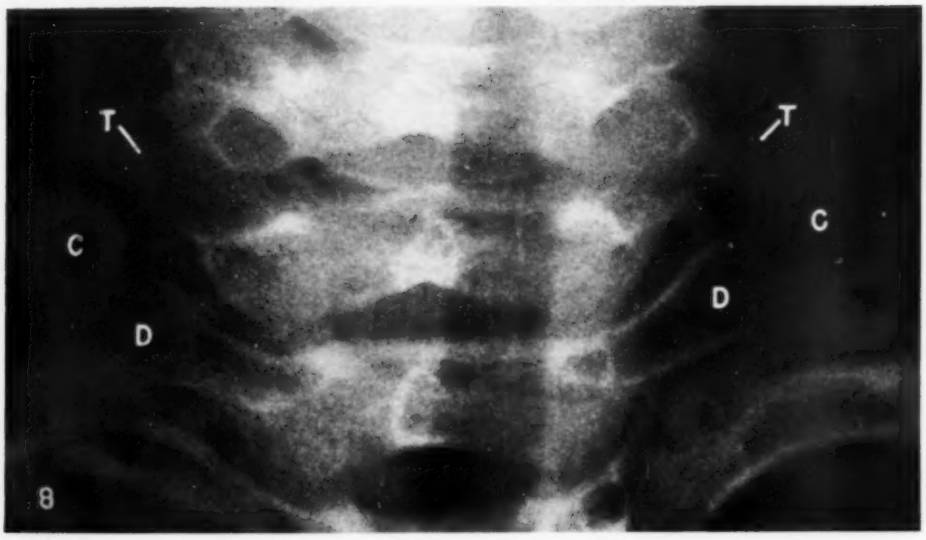


Figs. 4–6. Case 1. Fig. 4. Age 7 years. Prominent normal costal processes (C). Possible erroneous diagnosis of rudimentary cervical ribs. T. Transverse processes.

Fig. 5. Age 9 years. The costal processes (C), and transverse processes (T) are fusing. Note that the transverse process has increased in mass, while the costal process has maintained its original relative size.

Fig. 6. Age 13 years. Costal and transverse processes have now formed normal lateral masses and can no longer be easily distinguished. Cervical ribs have not developed.





Figs. 7 and 8. Case 2. Fig. 7. Age 2 years. Prominent costal processes (C) are within normal limits. An erroneous impression of cervical ribs might be created, especially on the left.

Fig. 8. Age 12 years. The costal processes (C) and transverse processes (T) are fused. There is slight asymmetry of the lateral masses thus formed, but no cervical rib has developed on either side. D. Transverse processes of D-1.

include an example of cervical ribs identified in the first few days of life, but we have observed them as early as six weeks of age. The growth of cervical ribs is thereafter only in proportion to growth of the normal thoracic ribs (Cases 4 and 5). The relatively static growth pattern exhibited by costal processes is thus duplicated by the growth pattern of cervical ribs.

With reference to the accurate diagnosis of cervical ribs, it is necessary to include two additional sources of possible error. The first is anomalous development of one or both first thoracic ribs. The second is the occasional presence of an extra cervical, dorsal, or lumbar vertebra, and the coexistence of thirteen more or less normally formed pairs of ribs. The latter situation is not common, but when present it can prove confusing.

In so far as is known, none of the patients whose films are reproduced here had symptoms clinically suggestive of cervical ribs.

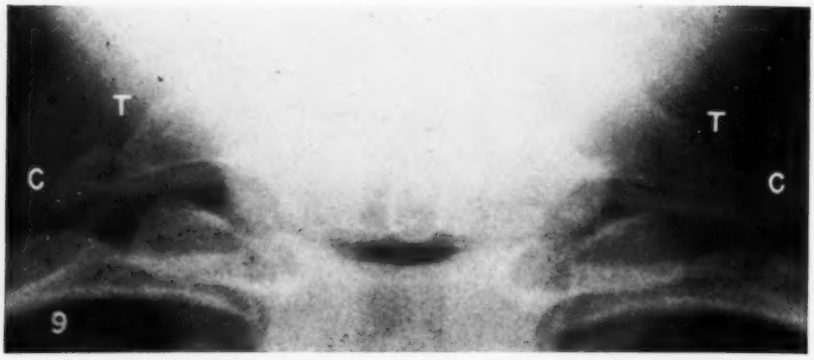
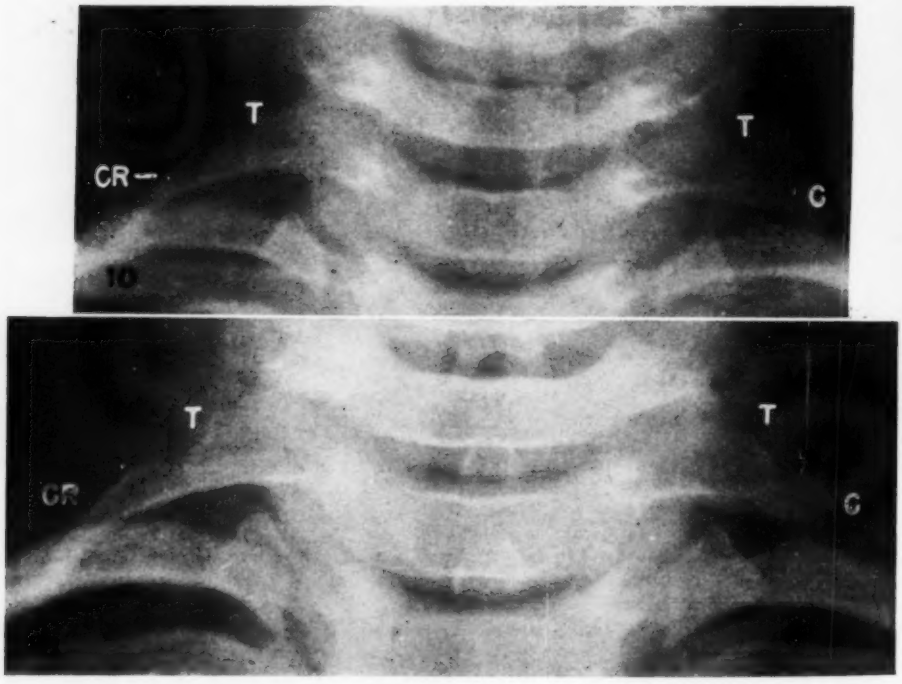


Fig. 9. Case 3. Age 9 years. Costal processes (C) more prominent than in Cases 1 and 2, tending to curve downward in the manner of true ribs, especially on the right. On both sides there is delayed fusion of costal and transverse processes. An arbitrary diagnosis of rudimentary cervical ribs is believed justified, but well formed cervical ribs will not develop.

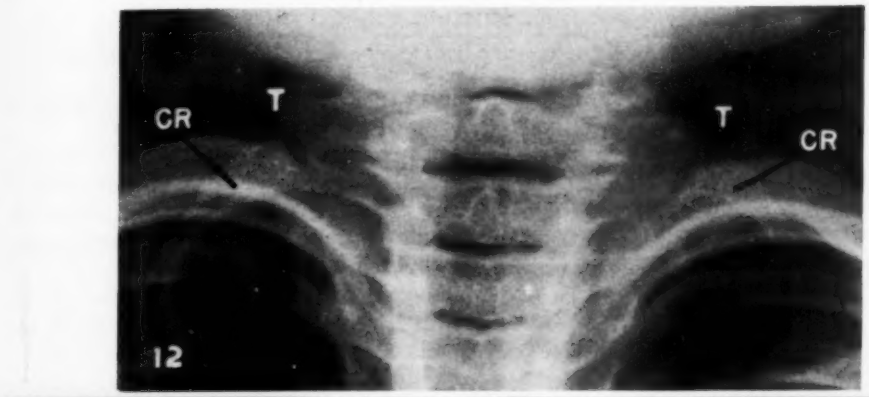


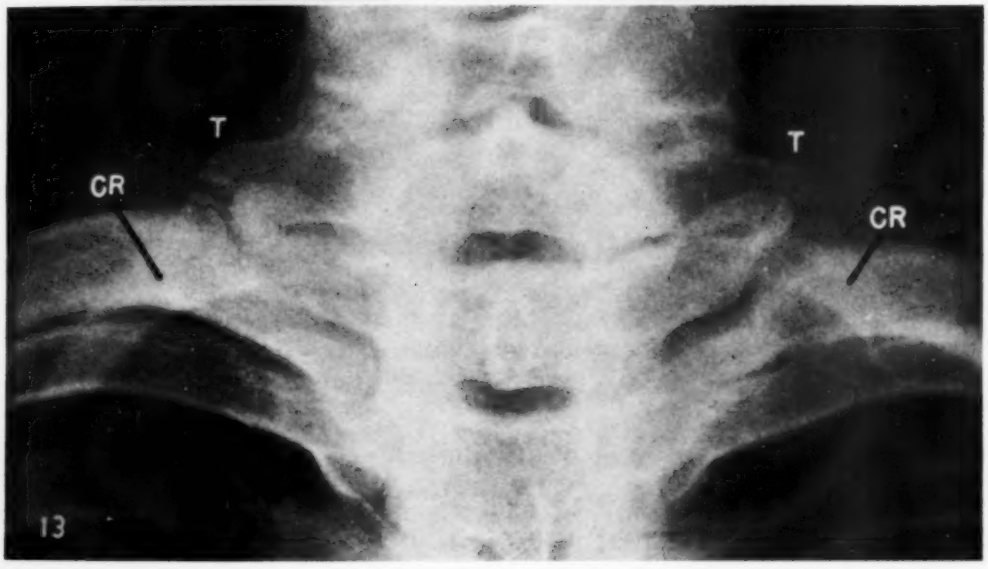
Figs. 10 and 11. Case 4. Fig. 10. Age 1 year. A rudimentary cervical rib (CR) replaces the right costal process from which the rib is formed. Normal, though prominent, left costal process (C). T. Transverse processes.

Fig. 11. Age 4 years. The rudimentary right cervical rib (CR) and the left costal process (C) have maintained their relative sizes in proportion to thoracic rib growth.

SUMMARY AND CONCLUSIONS

1. In childhood, the costal processes of the seventh cervical vertebra are independent of the transverse processes, and the costal processes may be so conspicuous in routine chest films as to result in an erroneous diagnosis of cervical ribs. The costal and transverse processes undergo





Figs. 12 and 13. Case 5. Fig. 12. Age 5 years. Bilateral rudimentary cervical ribs (CR) replace the costal processes from which the cervical ribs are formed. T. Transverse processes.
Fig. 13. Age 16 years. The cervical ribs have maintained growth relationship with each other and with the thoracic cage. Each cervical rib articulates with the transverse process of C-7 in the manner of normal thoracic ribs.

fusion at some time between the fourth and tenth years.

2. The costal processes of the seventh cervical vertebra are identical in embryogenesis with cervical and thoracic ribs.

3. Cervical ribs, when present, exist as such at birth, and whether they are rudimentary or are well formed they maintain their original size relationship to the thoracic ribs throughout the period of growth.

4. Normal costal processes of the seventh cervical vertebra in childhood are not potential cervical ribs.

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SUMARIO

Causa de Error Potencial en el Diagnóstico Roentgenológico de Costillas Cervicales

En la niñez, las apófisis costales de la séptima vértebra cervical son independientes de las transversas, y pueden destacarse tanto las primeras en las radiografías torácicas corrientes que conduzcan a un diagnóstico erróneo de costillas cervicales. Las apófisis costales y transversas experimentan fusión en alguna fecha entre el cuarto y el décimo años de vida. Las apófisis costales de la séptima vértebra cervical son idénticas en su embriogenia a las costillas cervicales y las dorsales.

Las costillas cervicales, cuando las hay, existen en esa forma desde el nacimiento y mantienen su primitiva proporción de tamaño con las costillas dorsales durante todo el período del desarrollo.

Las apófisis costales normales de la séptima vértebra cervical en la niñez no son potenciales costillas cervicales.

Myelography in Spinal Metastases[1]

SAUL HEISER, M.D., and ALFRED J. SWYER, M.D.

DURING THE PAST five years a total of 75 tumors involving the spinal canal have been studied by myelography at Montefiore Hospital. Twenty (27 per cent) of these proved to be metastatic cancer and 6 (8 per cent) lymphoma, a total of 26 (35 per cent).

The relatively high incidence of malignant tumors in this series can be explained by the fact that the institution from which this report emanates has a large, active neoplastic service, and patients are maintained under surveillance for the duration of their illness. The experience of others indicates a much lower frequency of spinal metastatic tumors. Sachs (15) found 20 (9 per cent) metastatic carcinomas and 28 (12 per cent) sarcomas (no further specification as to type) in a total of 234 spinal tumors. Elsberg (9) encountered only 14 (5 per cent) metastatic tumors in 267 cases. Wood (20) indicates the incidence to be 10 per cent. However, he makes no mention of lymphoma. Lymphomas of the spinal canal are less frequently encountered than would be implied by our statistics. Small collections of cases of lymphosarcoma (total of 20) have been reported in the past by Davison and Michaels (7), Browder and deVeer (4), Drake (8), Verda (18) and Shenkin, Horn and Grant (16). In a recent article on cord compression by Hodgkin's disease, by Smith and Stenstrom (17), the total number of cases of this disease with involvement of the central nervous system described in the literature was brought to about 66.

PATHOLOGY

Several mechanisms have been described to explain the occurrence of metastatic lesions in the epidural space of the spinal canal.

1. Hematogenous Spread: Tumor may seed in the spine *via* the arterial system. A primary pulmonary tumor may embolize directly into a pulmonary vein, avoiding the capillary bed. This holds true, also, for tumors which have metastasized to the lung and thus set up foci for further dissemination. Controversy exists regarding the ability of metastatic cells passing through the right side of the heart to filter through the lung.

2. Vertebral Vein System: The role of the vertebral veins in metastatic processes involving the spine was described initially by Batson (1, 2). This auxiliary system consists of a collection of valveless veins producing a rich anastomosis about the pelvic region, the vertebral column, and the cranium. The blood is carried through this system under low pressure, and the flow may be subject to arrest and even reversal in direction away from the heart. Batson showed experimentally in human cadavers and monkeys that, with increased intra-abdominal pressure, the venous blood flow from the pelvis and abdomen would not be through the portocaval channels but rather through the vertebral system of veins. Coman and deLong (6), experimenting with the injection of tumor cell suspensions (Walker rat carcinoma 256, and V2 carcinoma) into the femoral veins of rats and rabbits, reported a high incidence of spine involvement as compared to pulmonary metastases when abdominal compression had been applied.

The vascular needs of the spinal cord and subdural portion of the meninges are supplied by very fine vessels running intradurally along the nerve roots. These communicate with the vertebral vein system outside of the spinal column by entering and emerging at right angles through the perineurium. The sparing of the in-

[1] From the Department of Diagnostic Roentgenology, Montefiore Hospital, New York, N. Y. Presented before the New York Roentgen Society, April 20, 1953. Accepted for publication in June 1953.

tradural and cord regions from metastases may be explained on this basis

3. Extension of Tumor from Adjacent Osseous Structures: The high incidence of vertebral involvement in metastatic tumors is well known. This may be explained by hematogenous spread or extension by way of the vertebral veins, as described above. The nidus of metastatic tumor within the medullary portion of the vertebral body or neural arch may extend through the cortex into the epidural space or into the paravertebral soft tissues. Even without extra-osseous extension, severe destruction of the vertebral bodies may lead to collapse, subluxation, or angulation, with mechanical cord compression.

4. Direct Extension to Epidural Space via the Intervertebral Foramina: Tumor in the paraspinal soft tissues or in lymph nodes of the cervical, mediastinal, or retroperitoneal areas may invade the epidural space either directly or along the lymphatic channels accompanying the nerve roots.

5. Primary Growth of Lymphoma Within the Spinal Canal: Elements of the reticuloendothelial system in the epidural space may be the site of origin of a lymphoma. Indeed, lymphoma may arise in and be limited to the central nervous system. Three such examples are included in a report on malignant lymphoma within the central nervous system by Berkman, Netsky and Zimmerman (3).

A minimal epidural mass without mechanically compressing the cord can compromise the vascular supply, resulting in myelomalacia. Early relief of pressure will reduce the ischemia and possibly reverse the neurological changes. Prolonged unremitting pressure on the cord results in demyelinization and gliosis. This damage is permanent and irreversible.

The early pressure effects on the spinal cord are frequently on the pyramidal tracts. This is to be explained, according to Kahn (13), by the attachment of the dentate ligaments to the lateral segments of the cord, with resultant transmission of stress to the adjacent motor tracts.

CLINICAL AND LABORATORY DATA

The presenting symptoms and signs indicating the presence of an epidural mass may be pain due to nerve root compression, weakness of one or more extremities as a result of pyramidal tract involvement, or sensory disturbances caused by spinothalamic tract impairment. Sphincter disturbances may occur at any stage of cord or root compression.

In this series a correct clinical diagnosis of metastatic carcinoma or lymphoma was made in 15 cases. In the remaining 11, the diagnoses offered prior to myelography and surgery were primary cord tumor, sciatic syndrome with disk protrusion, virus myelitis, and ascending polyneuritis. In 3 of the 6 lymphoma cases, the correct diagnosis was not established prior to the laminectomy. Twenty of the 26 cases were histologically proved by laminectomy or autopsy. The remaining diagnoses were based upon the presence of a known primary tumor and the association of metastatic bone disease at the level of the neurological signs or response to irradiation in a known lymphoma. The primary source of tumor in the 20 cases of metastatic cancer were: lung, 5; breast, 3; prostate, 2; melanoma, 2; unknown, 2; kidney, uvula, thyroid, gallbladder, stomach, and tonsil, 1 each. These tumors were located as follows: cervical region, 3; dorsal, 8; lumbar, 8. The lymphomas, of which 4 were diagnosed as Hodgkin's disease and 2 as lymphosarcoma, were evenly divided, as to localization, between the dorsal and lumbar areas.

The cerebrospinal fluid pressures were lower than normal except in a few instances where the block was incomplete. Cerebrospinal fluid protein values ranged from 53 to 1,580 mg., generally depending on the degree of obstruction.

ROENTGENOGRAPHIC FINDINGS

Routine x-ray studies of the spine prior to myelography disclosed evidence of osseous metastases in 12 of the 20 cases of metastatic cancer. In 10 instances the

osteolytic or osteoblastic changes coincided with the level of cord compression. None of the 6 lymphoma cases presented any abnormalities of the vertebral osseous structures. Involvement of the skeletal system by Hodgkin's disease is infrequent. Falconer and Leonard (11) reviewed x-ray studies made on 750 cases and found bone lesions in 14.9 per cent. Two hundred and thirteen of these cases came to autopsy and 53.2 per cent showed anatomical evidence of bone involvement. Mazet (14), reporting on 28 cases of Hodgkin's disease involving the spine, found tumor present by radiographic as against autopsy examination in 17.8 and 71.4 per cent respectively.

The interpediculate measurements are generally not useful in the diagnoses of the lesions under discussion. Metastatic bone involvement results in either erosion or destruction of the roots of the neural arches. In contrast to this, slow growing tumors such as neurinomas or ependymomas cause thinning or displacement of the cortical margins because of prolonged extrinsic pressure. A similar statement may be made as to the value of determinations of the intervertebral foramina size. None of the tumors in this series showed evidence of calcification. This is a rare phenomenon usually associated with psammomatous meningiomas. Soft-tissue paravertebral masses were not observed.

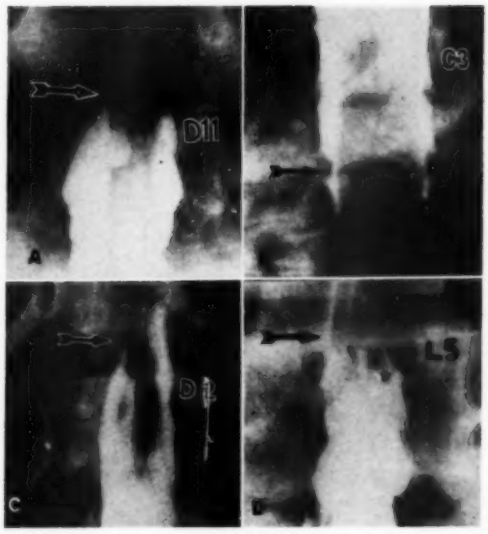

Fig. 1. Complete arrest of oil column by metastatic epidural tumor.

A. Metastasis from thyroid. Maximum extension of the oil column is at the lateral margins of the spinal canal. Central defect due to mid-line position of cord and tumor.

B. Metastasis from lung. Long, smooth central defect simulates an intramedullary tumor.

C. Metastasis from lung. Displacement of Pantopaque to the left indicates bulk of tumor on the contralateral side.

D. Hodgkin's disease. Large mass in lumbar region abruptly halts the contrast agent. Dentate appearance is caused by swelling of the nerve roots.

MYELOGRAPHIC FINDINGS

The defects found in the myelographic studies varied considerably. They depended on the degree of obstruction produced by the tumor, the configuration of the neoplasm, the relationship of the mass to the cord or, in the lumbar region, to the cauda equina, and the status of the supporting osseous structures.

1. Complete Block: In 18 of the cases there was a complete arrest of the oil column. In the cervicodorsal region, an obstructing mass not displacing the cord to either side was indicated by a rather smooth concave margin of the contrast medium. The mid-line position of the cord may be due to uniform annular peridural tumor infiltration or to a relatively small mass located either in the ventral or dorsal segment of the epidural space, compressing and angulating the cord. The maximum density and extension of the oil occurred in these instances along the periphery of the spinal canal (Fig. 1A and B). A growth predominantly unilateral caused deviation of the Pantopaque column to the opposite side (Fig. 1C). In one case the large size of the central defect, with minimal fusiform distention of the spinal canal, simulated an intramedullary lesion (Fig. 1B).

In the lumbar region the obstruction was usually more abrupt, with margins dentate (Fig. 1D). Absence of the cord here allows a large potential space for tumor growth. The dentate appearance might be accounted for by swelling and

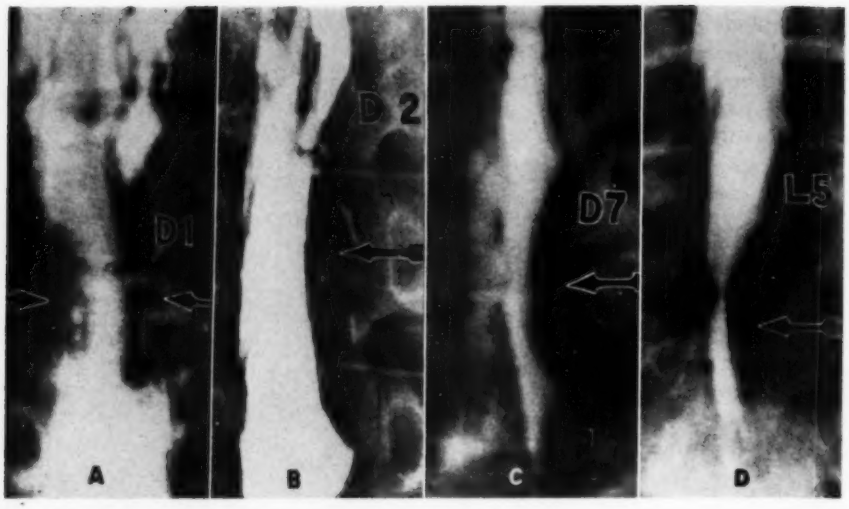

Fig. 2. Incomplete obstruction caused by epidural tumor.
A. Malignant melanoma. Bilateral pressure symmetrically constricts the subarachnoid space.
B. Lymphosarcoma.
C and D. Metastasis from prostate and lung respectively. Smooth lengthy tapering deformities extend over several vertebral segments.

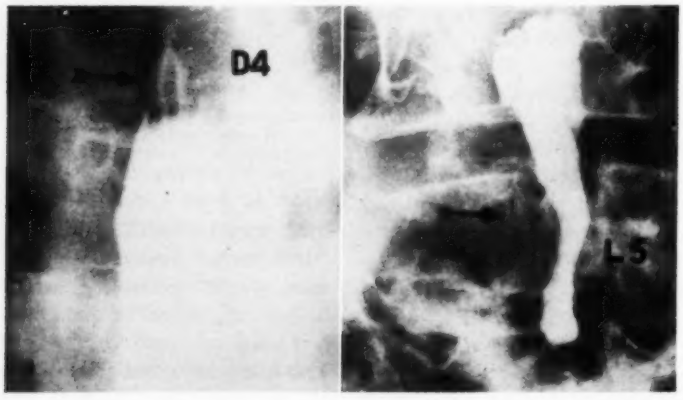

Fig. 3. Two spinal metastatic foci of adenocarcinoma (primary unknown) in the same patient. Absence of the cord in the lumbar region and destruction of the right half of L-5 account for the difference in myelographic appearance of the identical lesions.

distortion of the nerve roots of the cauda equina.

2. Partial Block: In the presence of partial block the defects consisted of unilateral, broad, smooth, sweeping indentations of the oil column, sometimes extending over several vertebral segments (Fig. 2). Occasionally the lesion was outlined as a series of convexities resembling a scalloped border (Fig. 2C). This appearance may be explained on the basis of a relatively soft tumor which is resisted by the cord, or by peculiarities of the growth itself, so that the lesion extends parallel to the cord or cauda equina rather than circumscribing it. In the lower lumbar spine, the asymmetrical growth of tumor may produce a gradual tapering of the oil column so that it resembles an irregular triangle with the apex down. The

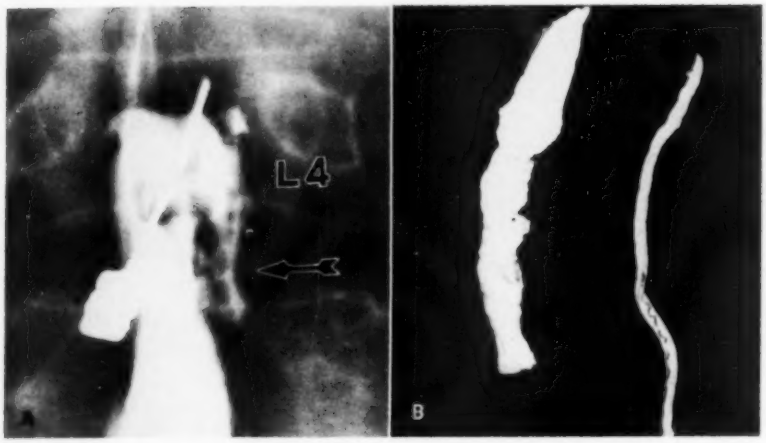

Fig. 4. Intradural metastasis to cauda equina from lung adenocarcinoma.
A. Defects produced by the large nerve roots.
B. Photograph of one of the resected nerves infiltrated by tumor (left). Compare with caliber of a normal root (right).

extreme caudal end of the subarachnoid space may be smoothly cut off (Fig. 2D). There were only 2 instances of this type of defect in the 10 cases of this series where the tumor occurred at the level of L-4–5 or below. This is contrary to the experience of Epstein (10), who recently reported such a configuration in 7 of his 11 cases of epidural metastases of the lumbosacral portion of the spinal canal. In a single instance (Fig. 2A) the pressure defect was bilateral, causing considerable constriction of the spinal canal. Further annular peridural tumor infiltration would have led to an appearance of complete arrest.

Multiple intraspinal metastatic lesions were demonstrated in only 1 patient (Fig. 3). The dissimilarity in the myelographic distortion produced by identical focal metastatic neoplasms could be accounted for by the difference in location of the two tumors and the added factor of bone involvement with lumbar scoliosis.

In only 1 patient was the tumor found to be intradural in location. Myelograms showed thickening and distortion of the cauda equina nerve roots (Fig. 4A). This was seen on resection of the lesion to be due to infiltration of the nerves by tumor (Fig. 4B).

The 6 cases of lymphoma involving the spinal epidural space resulted in various types of myelographic defects. There was no basis on which to separate this group from the metastatic carcinomas.

DIFFERENTIAL DIAGNOSIS

Intramedullary tumors, usually gliomas, generally present a configuration so characteristic that little confusion is encountered in differentiating these from extradural lesions. Intradural, extramedullary tumors (predominantly meningiomas or perineurial fibroblastomas) are generally more abruptly and sharply demarcated because of the absence of intervening dura between the tumor and contrast agent (Fig. 5A). As described by Camp (5), "the margin of the tumor that projects against the contrast medium will be depicted by a clearcut crescentic or concave defect." The tumor appears to be "capped" by the contrast oil. In reviewing the myelograms of all of the intradural, extramedullary tumors, this so-called typical appearance was found to hold true in less than half of the cases (Fig. 6A and B). Thus, differentiation between benign intradural and malignant extradural lesions may at times be impossible. This may be explained by the development of arach-

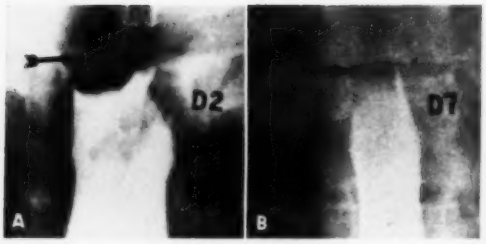

Fig. 5. Obstruction produced by perineurial fibroblastomas.

A. Extramedullary, intradural location. Margin of tumor is sharply outlined by concave indentation of the oil column, considered typical of a mass in this location. Cord is displaced to the left.

B. Extradural location. Defect produced similar to defects associated with other extradural lesions. Compare with Fig. 1.

noidal adhesions, causing such distortion of the oil column as to resemble the irregularities of some extradural malignant growths. Any epidural lesion may simulate the myelographic appearance of metastatic tumors (Fig. 5B). In the lumbosacral area, large herniated intervertebral disks especially may offer problems in differential diagnosis (Fig. 6C and D). Small disk protrusions producing unilateral or central smooth defects and obliteration of the nerve root sleeves are generally sufficiently characteristic so that there is no difficulty in distinguishing these from other tumors.

The long plaque-like unilateral defects observed in the dorsal area only are probably most characteristic of metastatic tumors. However, an unusual and infrequently seen lesion such as an extradural granuloma, fibrolipoma, or hemangioma may produce a similar pattern.

DISCUSSION

The first sign of metastasis or even the first indication of the presence of a malignant tumor may be due to spinal root compression. The symptoms of root involvement often point to an intrathoracic or abdominal organ as the offender. With this in mind, numerous radiographic and laboratory studies may be done without uncovering an organic disease. A spinal film may give the clue to the origin of the symptoms. If this is unrevealing, serious

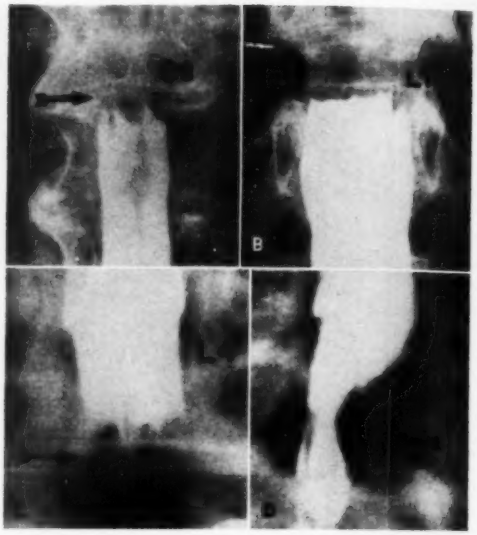

Fig. 6. Examples of primary intradural and extradural lesions to be differentiated from metastatic tumors.

A. Extramedullary intradural perineurial fibroblastoma. Contrast this configuration with so-called typical deformity of tumor in this location shown in Fig. 5A.

B. Extramedullary intradural meningioma.

C. and D. Epidural herniated intervertebral disks. Compare D with Fig. 2D.

consideration should be given to myelography.

Even in the presence of a known primary tumor one should not assume that the development of neurological symptoms and signs is necessarily due to a metastatic focus. Freedman, Feiring and Davidoff (12) reported 3 cases with carcinoma of the breast in which intraspinal meningiomas were discovered by surgical laminectomy. In 2 of the cases mastectomy had been performed five years previously. In the third case carcinoma of the breast with axillary metastasis was found on physical examination following admission to the hospital for paraplegia. One month after radical mastectomy, laminectomy disclosed an intradural psammomatous meningioma.

As soon as spinal cord or nerve root pressure is suspected, an emergency situation exists, since prolonged pressure on these nerve tissues produces irreversible organic changes. Therefore, an immedi-

ate investigation for tumor should be undertaken. In the presence of a known primary lesion but without evidence of spread elsewhere, or even with extensive metastasis but a reasonable prognosis, myelography and laminectomy should be done. In the case of a radiosensitive tumor, irradiation may be substituted, providing the danger to the spinal cord due to reaction is not too great.

Multiplicity of intraspinal lesions can be demonstrated only by myelography. The neurological signs and symptoms alone are not sufficient to distinguish single from multiple spinal tumors. It may be necessary to do myelography *via* the lumbar and cisternal routes to outline both tumors or the complete extent of a single lesion which is producing complete obstruction.

Caution should be exercised during myelography in the presence of complete obstruction. The removal of excess fluid may result in a downward shift of the tumor, producing a physiological transection of the cord due to further interference with cord circulation. It is far safer to instill Pantopaque after removal of a small amount of fluid and not attempt to remove it at the end of the procedure. Instillation of the medium by the cisternal route is not associated with this risk.

CONCLUSION

The myelographic findings in 26 cases of metastatic tumors and lymphomas are presented. In most instances the defects found are not specific, and differentiation from other extradural and even intradural lesions may be difficult. Metastasis to the spine may be the first evidence of malignant disease. The signs and symptoms produced particularly by nerve root compression may simulate intrathoracic or intra-abdominal lesions, and the attention of the physician may be erroneously focused on these areas. Early myelography and laminectomy are urged when there is clinical evidence of cord compression even in the presence of a known primary tumor.

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SUMARIO

La Mielografía en las Metástasis Raquídeas

Un total de 75 tumores que afectaban el conducto raquídeo fué estudiado con la mielografía, resultando ser 20 de ellos metástasis carcinomatosas y 6 linfomatosas. Este grupo de 26 forma la base de este trabajo.

La radiografía corriente del raquis reveló signos de metástasis óseas en 12 de los 20 casos de carcinoma, pero no mostró la menor anomalía de los tejidos vertebrales en los 6 casos de linfoma.

Los hallazgos mielográficos variaron, de acuerdo con la gravedad de la oclusión producida por el tumor, su configuración, su relación con la médula o la cola de caballo y el estado de los tejidos óseos de apoyo. En la mayor parte de los casos, los defectos observables mielográficamente que acompañan a esos tumores no son específicos y tal vez resulte difícil la diferenciación de otras lesiones extradurales y hasta intradurales.

Las metástasis en el raquis quizás sean el primer signo de una afección maligna. Los signos y síntomas producidos en particular por la compresión de las raíces nerviosas pueden simular lesiones intratorácicas o intraabdominales, y el médico acaso concentre su atención erróneamente en dichas zonas. Recomiéndanse la mielografía y la laminectomía tempranas cuando existen signos clínicos de compresión de la médula, aun conociéndose la existencia de un tumor primario.

Roentgenographic Demonstration of Histologically Identifiable Renal Calcification[1]

JD MORTENSEN, M.D.,[2] ARCHIE H. BAGGENSTOSS, M.D.,[3] MARSCHELLE H. POWER, Ph.D.,[4] and DAVID G. PUGH, M.D.[5]

DEPOSITION of calcium in renal parenchyma is a common pathologic finding. Frequently it is observed in kidneys which have functioned normally and in which there are no other recognizable histologic abnormalities. Its clinical significance, therefore, is at present poorly understood. On the other hand, although it is of rare occurrence, clinically recognizable (that is, roentgenographically demonstrable) renal calcification, known generally as "nephrocalcinosis," has clinical implications of importance. The relationship of commonly observed but questionably significant renal depositions of calcium, found pathologically, to the rare but evidently significant renal calcification which is demonstrable roentgenographically thus becomes a matter of interest and importance.

Commonly, and perhaps logically, renal calcification has been considered a rather specific lesion, even though it may be brought about by several different processes and may be controlled by various unrelated mechanisms. Aside from classification into the two subgroups, metastatic and dystrophic, little interest has been shown in subdividing cases of renal calcification into more homogeneous groups. With the advent and wide use of clinical roentgenography of the kidneys, an important practical method of recognizing certain types or degrees of renal calcification became available. Before applying all the accumulated knowledge concerning pathologically demonstrable renal calcification to those patients in whom such calcification is recognizable roentgenographically, it would appear essential to make sure that the two groups represent the same process. On the other hand, if differences exist between pathologically identified renal calcification and roentgenographically demonstrable nephrocalcinosis, those differences should be known. Indeed, they might prove to be useful clinically even though they ultimately may be shown to represent different stages of the same process.

In order to investigate the possible relationships between renal parenchymal calcification that is recognized histologically only and renal parenchymal calcification that can be identified on the roentgenogram, the following study was undertaken.

MATERIALS AND METHODS OF STUDY

From the files of the Section of Pathologic Anatomy at the Mayo Clinic all cases were selected in which routine histologic study of renal tissue revealed calcium deposition of sufficient degree that the case had been indexed under a diagnosis of renal parenchymal calcification. From this large number of cases were chosen all those in which gross specimens of the kidneys were available and intact, in a condition suitable for roentgen examination. In this manner it was possible to obtain for roentgenographic study one or both kidneys from 210 cases; all showed renal calcification which could be identified histologically. No kidney which met these qualifications was omitted from the study.

The histologic sections of each specimen were reviewed and the presence of renal calcification was verified. Each kidney was then subjected to roentgen examination by technics which previously had been determined to be most efficient and prac-

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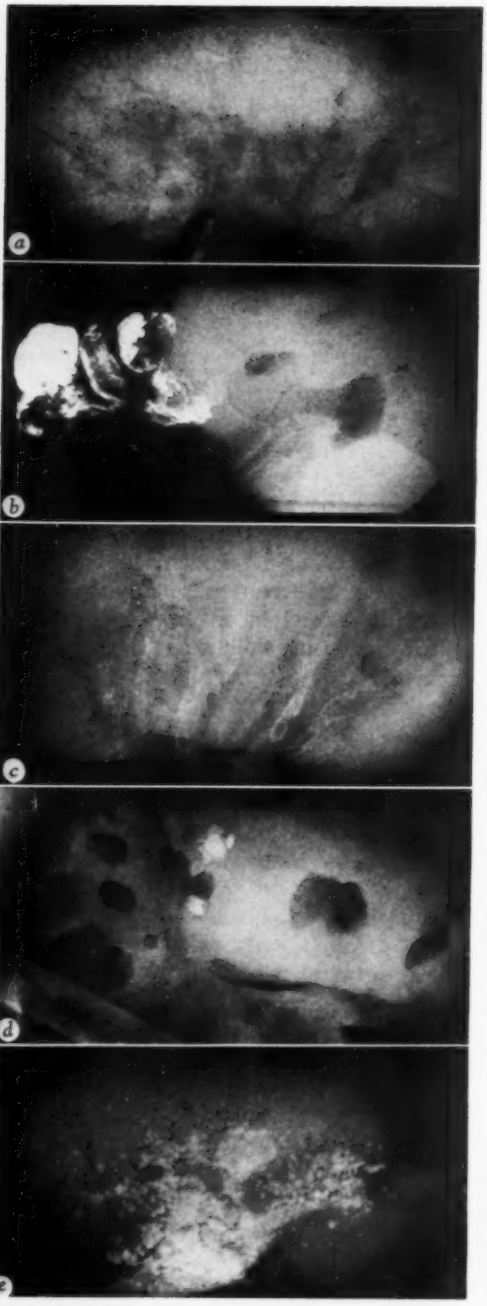

Fig. 1. Postmortem roentgenograms of kidneys containing histologic evidence of calcification in the parenchyma which was not demonstrable roentgenographically. (*a*) Although calcium depositions were present, they were not evident roentgenographically. (*b*) Calcification of an old infarct, but no evidence of diffuse deposits of calcium in the renal parenchyma. (*c*) Calcified renal vessels in a patient with chronic glomerulonephritis. (*d*) Discrete renal calculi, not nephrocalcinosis. (*e*) Calcium-containing deposits in calyces and pelvis of kidney from patient with multiple myeloma.

TABLE I: POSTMORTEM ROENTGENOGRAPHIC EXAMINATION OF KIDNEYS FROM 210 CASES WITH HISTOLOGICALLY EVIDENT RENAL CALCIFICATION

Roentgenographic Finding		Number of Cases
No roentgenographic evidence of calcium deposition		174 (82.8%)
Localized calcium deposition		30 (14.3%)
Deposits in an isolated pathologic lesion (cysts, neoplasm, infarct)	16	
Calcified renal vessels	4	
Discrete renal calculus	5	
Calculi in calyces or pelves	5	
Multiple diffuse parenchymal calcifications (nephrocalcinosis)		6 (2.9%)
TOTAL		210 (100%)

tical for demonstrating minute deposits of calcium in isolated kidneys. This involved placing the kidney directly on a cardboard holder containing non-screen film, the specimen being 42 inches from the roentgen tube. Exposure was made at 49 kv.p. at 22 to 37 ma. seconds, depending on the thickness of the specimen.

The roentgenograms thus obtained were studied carefully, special note being made of evidence of radiopaque shadows of any nature or distribution. After the roentgenograms had been interpreted, the clinical record of each patient was reviewed, as well as all findings on complete postmortem examination.

The remainder of the study involved special technics which were limited to selected cases for the purpose of investigating various possible explanations for the overall findings. These special studies are described below, under the heading, *Auxiliary Preliminary Investigations.*

RESULTS

Roentgenographic Findings: Of the 210 cases in which kidneys were found to contain histologic evidence of calcification, 174 (82.8 per cent) did not show radiopaque shadows of any type (Table I; Fig. 1*a*). In 30 cases (14.3 per cent) radiopaque shadows were evident in iso-

lated portions of the kidney or in localized pathologic lesions involving the kidney (Fig. 1*b*, *c*, *d*, *e*). In 16 of the 30 cases, the calcification seen roentgenographically consisted simply of local deposits of calcium in isolated areas where some local pathologic lesion was found, such as an infarct (Fig. 1*b*), cyst, abscess, neoplasm, or the like. Calcified renal vessels (Fig. 1*c*) were recognized roentgenographically in 4 cases. Discrete calculi in the renal parenchyma were identified in 5 cases (Fig. 1*d*) and stones were noted in the calyceal system or renal pelves in 5 cases (Fig. 1*e*). Thus, in none of the 30 cases could the roentgenographic manifestation in any way be considered to represent the histologically evident calcification in the renal parenchyma. In only 6 cases (2.9 per cent) was nephrocalcinosis demonstrated roentgenographically, and in all 6 cases the roentgen manifestations were characteristic and easily identifiable (Fig. 2*a* and *b*).

On critical study of these 6 cases of nephrocalcinosis, it was found that the roentgenographic manifestations could be divided into two groups: those in which the deposits of calcium appeared to be limited to the medulla, concentrated primarily at the tips of the medullary pyramids (Fig. 2*a*), and those in which the deposits of calcium were scattered throughout the parenchyma, being equally intense in the cortex and in the medulla (Fig. 2*b*). Four of the 6 cases were included in the first group, corresponding to the usual roentgen picture of clinically recognizable nephrocalcinosis, while 2 cases represented a more unusual type, heretofore clinically recognized almost exclusively among patients having chronic glomerulonephritis (1, 2).

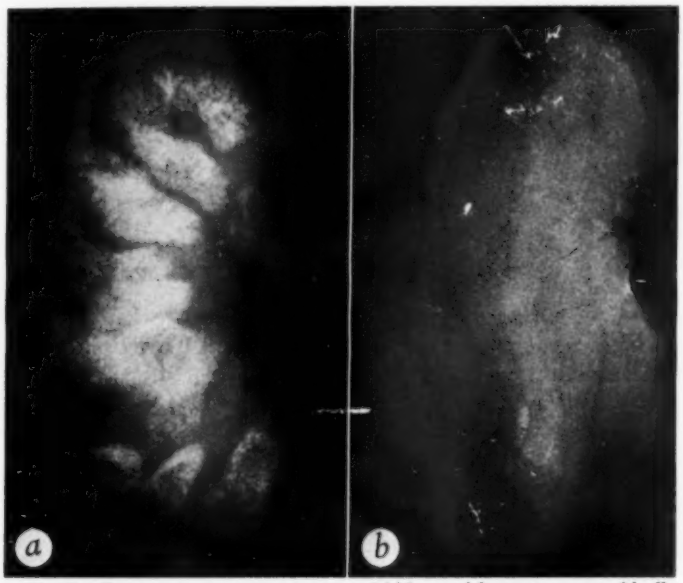

Fig. 2. Postmortem roentgenograms of kidney with roentgenographically demonstrable nephrocalcinosis. (*a*) Typical nephrocalcinosis with deposits of calcium in medullary pyramids. (*b*) Unusual manifestation of nephrocalcinosis with deposits of calcium scattered throughout entire renal parenchyma.

Auxiliary Preliminary Investigations: In most kidneys pathologically recognizable deposits of calcium in the parenchyma did not cast identifiable roentgenographic shadows. On the other hand, in a few kidneys histologically similar deposits of calcium did cast roentgenographic shadows characteristic of nephrocalcinosis. When these facts became evident, our interest was aroused concerning possible reasons for the

TABLE II: RELATIONSHIP BETWEEN GROSS ESTIMATION OF EXTENT OF RENAL CALCIFICATION AND ITS ROENTGENOGRAPHIC DEMONSTRABILITY

Roentgenographic Manifestation of Histologically Evident Calcium	Gritty Sensation on Cutting, (per cent of cases)	Gross Estimation of Extent of Calcification (per cent of cases)				
		No Visible Calcification	Grade 1	Grade 2	Grade 3	Grade 4
Not demonstrable roentgenographically, 174 cases	52.9	20.1	10.3	53.4	14.9	1.2
Demonstrable roentgenographically, 6 cases	83.0	0	33.3	16.7	33.3	16.7

TABLE III: RELATIONSHIP BETWEEN HISTOLOGIC ESTIMATION OF EXTENT OF RENAL CALCIFICATION AND ITS ROENTGENOGRAPHIC DEMONSTRABILITY

Roentgenographic Manifestation of Renal Calcification, 18 Cases	Calcium Deposits							
	Grade (by size)				Grade (by number of deposits)			
	1	2	3	4	1	2	3	4
Not recognizable roentgenographically, per cent of 12 cases	8	33	41	17	17	41	17	25
Recognizable roentgenographically, per cent of 6 cases	0	50	33	17	17	17	33	33

differences in roentgen demonstrability of these lesions. In an attempt to substantiate a few possible explanations, the following preliminary auxiliary investigations were carried out:

1. Gross Estimation of Quantity of Calcium Deposition: The most logical explanation of differences in roentgenographic demonstrability of renal parenchymal calcifications would seem to be the quantity of calcium salts deposited. In estimating how much calcium was deposited in the kidneys, each kidney was first studied grossly. On careful inspection of a freshly cut surface, we graded the amount of visible calcification, if any, from 1 (minimal degree) to 4 (maximal degree). In addition, we recorded whether or not a gritty sensation was encountered on cutting the renal substance. In Table II are recorded the results of this gross study of the kidneys from the 6 cases which showed roentgen evidence of nephrocalcinosis compared to the gross findings of the 174 kidneys with histologically evident, but not roentgenographically demonstrable, parenchymal calcification. Although in general there appeared to be slightly more calcium in the roentgenographically positive kidneys, the differences are not great, and we must conclude that gross estimation of the amount or degree of renal parenchymal calcification is not a reliable indication of whether or not that kidney will cast recognizable roentgenographic shadows characteristic of nephrocalcinosis.

2. Histologic Examination: In order to investigate further the importance of the quantity of calcium deposited, we studied several microscopic sections of renal tissue taken through areas in which deposits of calcium were densest in each of the 6 cases showing roentgenographically demonstrable nephrocalcinosis and in 12 cases selected at random from those which did not cast roentgenographic shadows. After careful histologic study, the slides of these 18 cases were graded according to number of deposits and again according to size of the largest deposits. The histologic classifications were done without knowledge of the roentgenographic findings. Results (Table III) indicate that histologic estimation of degree or quantity of calcium deposited in the renal parenchyma is not a reliable method of determining whether the calcium deposits will or will not cast recognizable roentgenographic shadows.

3. Studies with Differential Histologic Stains: Von Kossa's stain was used in the histologic study of sections from 20 kidneys: 6 with roentgenographically demonstrable nephrocalcinosis and 14 with histologically evident calcifications not

TABLE IV: RELATIONSHIP OF CAUSATIVE DISORDER TO ROENTGENOGRAPHIC DEMONSTRABILITY OF RENAL PARENCHYMAL CALCIFICATION

Evident Causative Disorder	No. Cases	Results of Roentgenographic Examination		
		No Evidence of Calcification	Localized Calcification	Nephrocalcinosis
I. Lesions capable of producing hypercalcemia or hypercalciuria or both				
Primary hyperparathyroidism	7	2	2	3
Osteolytic bone lesions				
Metastasis	20	18	2	0
Multiple myeloma	8	6	2	0
Diffuse osteoporosis	5	5	0	0
Vitamin D intoxication	1	1	0	0
II. Metabolic disorders predisposing to calcium precipitation without hypercalcemia				
Chronic peptic ulcer with hypochloremic alkalosis	4	4	0	0
Chronic peptic ulcer without demonstration of hypochloremic alkalosis	8	7	1	0
Recurrent "idiopathic" renal lithiasis	4	3	1	0
III. Local injury to renal tissue				
Acute pyelonephritis	2	2	0	0
Chronic pyelonephritis	2	1	0	1
Acute or subacute glomerulonephritis	2	2	0	0
Chronic glomerulonephritis	4	2	1	1
Renal tuberculosis	6	3	3	0
Hydronephrosis and pyonephrosis	4	4	0	0
Nephrosclerosis (benign and malignant)	8	7	1	0
Sulfonamide tubular damage	3	3	0	0
Mercuric poisoning	3	3	0	0
Polycystic kidneys	3	3	0	0
Local renal pathologic lesions (cysts, abscesses, infarcts, neoplasms)	20	8	12	0
Miscellaneous renal disorders (arsenic poisoning, compensatory hypertrophy, postoperative anuria, etc.	10	8	2	0
IV. Miscellaneous lesions with accompanying renal calcification from undetermined causes				
Multiglandular dysfunction with postoperative adrenal insufficiency	1	0	0	1
Other miscellaneous lesions	85	82	3	0
TOTAL	210	174	30	6

manifest on the roentgenogram. In all cases the deposits stained positively for calcium. It was not possible to estimate the density of the calcium deposits histologically, all appearing to stain to approximately the same intensity with routine hematoxylin and eosin stain as well as with the von Kossa silver technic. Since roentgenographically demonstrable cerebral deposits of calcium regularly contain significant quantities of iron (3), we undertook to identify iron with the use of Berlin blue stain in the sections of 3 kidneys showing nephrocalcinosis on the roentgenograms and 6 kidneys with parenchymal calcifications which did not appear roentgenographically. In none of these tissues were significant amounts of iron found.

Thus we were unable, by simple histologic staining technics, to establish differences in density or chemical composition which would explain why certain deposits of calcium in the renal parenchyma were roentgenographically demonstrable while others were not.

4. Histopathologic Characteristics: Although our results were not representable statistically, we attempted to formulate histopathological characteristics which would make it possible to distinguish kidneys with roentgenographically demonstrable nephrocalcinosis from those with histologically evident renal calcifications which did not appear roentgeno-

TABLE V: CORRELATION BETWEEN QUANTITATIVE CHEMICAL DETERMINATION OF CALCIUM CONTENT AND GROSS HISTOLOGIC AND ROENTGENOLOGIC IDENTIFICATION OF CALCIUM DEPOSITS IN 14 KIDNEYS

Case	Evidence of Calcification			Calcium, as Determined Chemically (gm. per 100 gm. dried tissue)
	Histologic	Gross	Roentgenographic	
1	0	0	No roentgenogram	0.30
2	0	0	No roentgenogram	0.21
3	++	+++	0	0.40
4	+++	++	0	0.43
5	++	+	0	0.24
6	+	0	0	0.39
7	++	0	0	0.24
8	+	++	0	0.27
9	++	+++	+++	1.47
10	+++	+++	++	1.66
11	+	++	++	1.77
12	++	++	+	0.40
13	++++	+	+	2.30
14	+++	+	++	0.53

graphically. We must report that we were unable to do so. In general, kidneys which contained roentgenographically demonstrable nephrocalcinosis gave histologic evidence of more inflammatory reaction and more replacement of renal parenchyma by fibrosis than was observed in most kidneys in which the calcification was not apparent roentgenographically. This was not invariably so, however, and it was impossible to predict, from the histopathologic features alone, which kidneys would be shown roentgenographically to contain calcification and which would not.

5. Associated Causative Disorders: It might be postulated that the renal calcification secondary to certain causative disorders could be more radiopaque than the calcification produced by other mechanisms. In order to appraise such a concept, we reviewed the clinical and pathologic findings in each of our 210 cases and recorded the disorder which appeared responsible for the production of the renal calcification. Results of this attempt to correlate the causative disorder with the roentgenographic demonstrability of the renal calcification indicate that primary hyperparathyroidism, chronic glomerulonephritis, and chronic pyelonephritis produce renal calcification that is likely to be roentgenographically demonstrable (Table IV). On the other hand, such disorders as hypochloremic alkalosis accompanying chronic duodenal ulcer, osteolytic lesions of various types, vitamin D intoxication, and renal insufficiency secondary to a variety of organic renal lesions commonly resulted in histologically identifiable renal calcification, but in no instance could the calcium deposits be identified roentgenographically. These results are consistent with accumulated clinical experience, according to which the only disorders yet demonstrated to play well established etiologic roles for clinically recognized nephrocalcinosis are primary hyperparathyroidism, hyperchloremic acidosis (Albright's tubular insufficiency without glomerular insufficiency), chronic pyelonephritis, chronic glomerulonephritis, recurrent "idiopathic" renal lithiasis, and "idiopathic" hypercalciuria (4, 5).

6. Quantitative Chemical Determinations: It is well known that certain renal calculi are radiolucent, while others are radiopaque. The presence and relative abundance of calcium in these stones appear to be factors of importance in this respect. By analogy, the roentgenographic demonstrability of histologically evident deposits of calcium in the renal parenchyma would be expected to depend to some extent on the concentration of calcium. Quantitative chemical analyses were therefore performed on renal tissue from 6 cases in which the calcium could be recognized roentgenologically, from 6 cases with histologically but not roentgenologically demonstrable calcium, and from 2 control cases without gross or histologic evidence of deposits of calcium. The results, as shown in Table V, indicate a positive correlation between the chemically determined quantity of calcium in renal tissue and its roentgenologic demonstrability. In kidneys without gross, histologic, or roentgenologic evidence of cal-

cification, calcium was present in a concentration of up to 0.2 gm. per 100 gm. of dry renal tissue. Concentrations of about 0.2 to 0.5 gm. of calcium per 100 gm. of dry tissue were commonly found in kidneys with grossly and histologically evident deposits of calcium which, however, were not apparent on the roentgenogram. All the kidneys with roentgenographically demonstrable nephrocalcinosis contained more than 0.4 gm. of calcium per 100 gm. of dry tissue and 4 of 6 nephrocalcinotic kidneys contained more than 1 per cent calcium by weight. It should be pointed out that in these quantitative chemical studies the results may be variable, depending on the amount and source of renal tissue submitted for analysis. We selected approximately 5 gm. of tissue from regions in which highest concen-

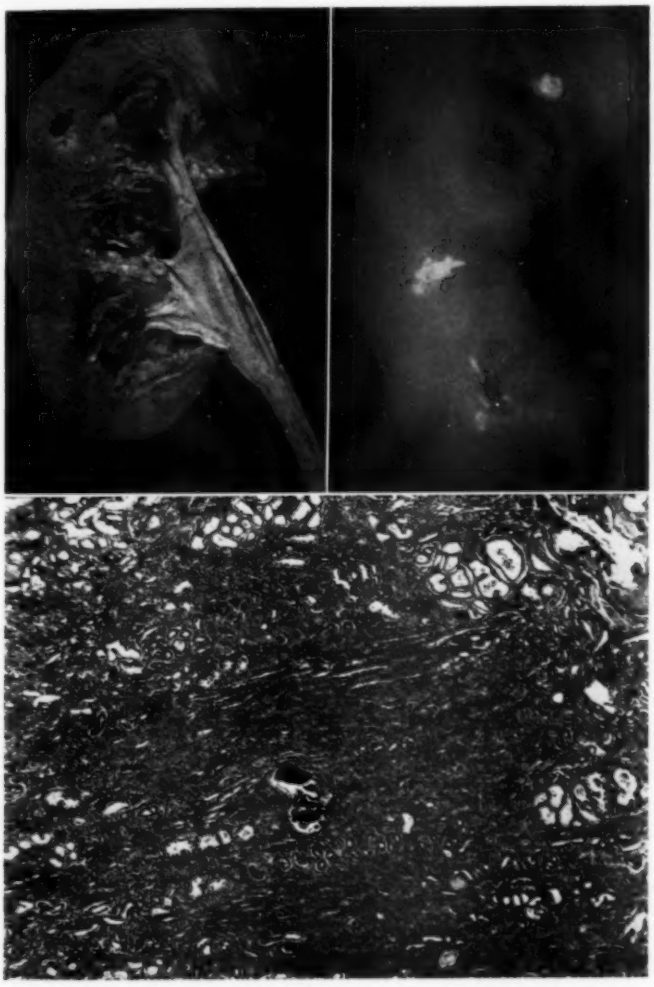

Fig. 3. Relationship of histologic, gross, and roentgenologic demonstration of renal calcification. This patient had primary hyperparathyroidism and resultant nephrocalcinosis. The correct diagnosis is not readily apparent on gross or histologic examination of the kidneys, but the roentgenogram demonstrates unmistakable evidence of nephrocalcinosis. The kidneys contained 1.77 gm. of calcium per 100 gm. dry kidney weight. Compare with Fig. 4.

trations of calcium were grossly evident.

We deduce from these preliminary studies that, although the quantity of calcium present in renal tissue, as determined by quantitative chemical analysis, appears to be directly related to the ability of that tissue to cast recognizable roentgenographic shadows, neither gross nor histologic estimations of the size, number, density, or extent of renal calcifications are reliable indications of their roentgenographic demonstrability (Figs. 3 and 4).

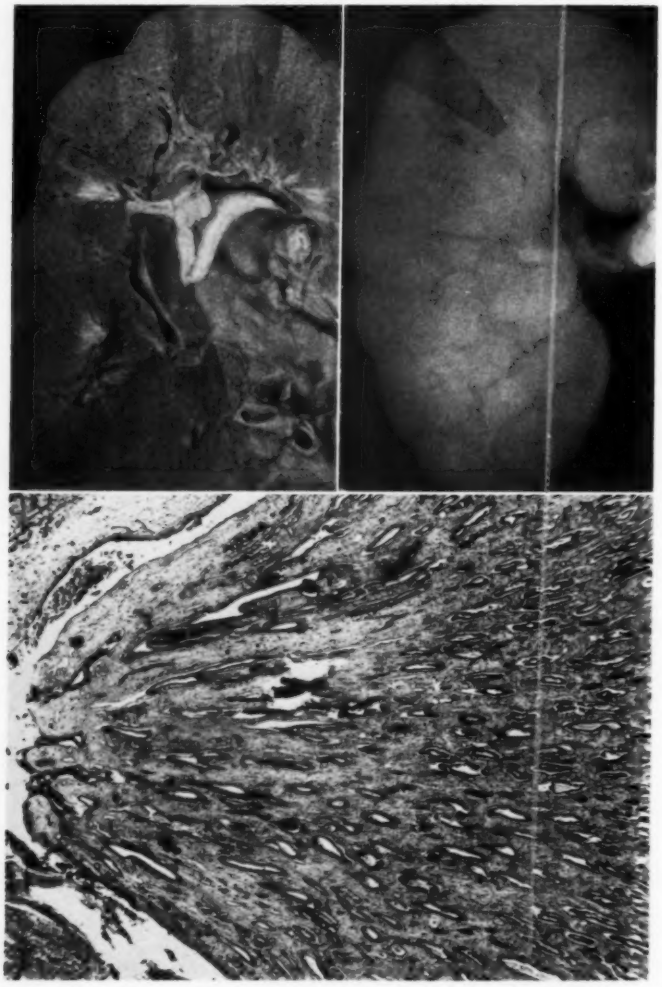

Fig. 4. Compare with Fig. 3. Relationship of histologic, gross, and roentgenologic demonstration of renal calcification. This patient was an 83-year-old diabetic who died with pneumonia and a fractured hip. The cause for renal calcification is not apparent. Note the easily identifiable deposits of calcium on histologic and gross examination; the roentgenogram, however, did not show evidence of calcification. The kidneys contained 0.4 gm. of calcium per 100 gm. dry kidney weight.

COMMENT

The data presented here appear to indicate rather conclusively that most of the renal parenchymal calcification recognized histopathologically is not demonstrable roentgenographically. This is not an unexpected finding, and it is in

accord with findings in other organs in which calcification is identifiable chemically or histologically much earlier than it can be recognized grossly or roentgenographically. The logical explanation for this lag between the histologic recognition of calcification and its roentgenographic demonstration is that the process must progress until the deposits become large enough, numerous enough, or dense enough to cast recognizable shadows on the roentgenogram.

The validity of this explanation is not questioned, but the preliminary auxiliary investigations which we conducted suggest to us that other factors may contribute importantly to the radiolucency or radiopacity of various renal parenchymal calcifications. We do not propose that these preliminary studies rule out the possibility that size, number, distribution, density, and chemical composition of the deposits are the important characteristics accounting for roentgenographic demonstrability, but we do suggest that such suppositions should be investigated and established by more accurate and definitive methods.

Similarly, we do not maintain that this study indicates that the diffuse calcification of the renal parenchyma which is demonstrable roentgenographically is biologically or basically different from renal calcification which is only recognizable histologically. So far as we know, such a supposition has not been investigated, and objective data are not available for either its support or negation. We do not believe, however, that it is accurate or justifiable to consider these lesions, which are demonstrable by different methods, as identical or in all particulars comparable until such relationships have been established.

Moreoever, it appears logical to us that clinically recognizable (that is, roentgenographically demonstrable) diffuse calcification of the renal parenchyma has different etiologic, diagnostic, prognostic, therapeutic, and pathologic implications than can be attached to histologically identifiable but not roentgenographically demonstrable renal calcification. For this reason, though the two types may ultimately be found to represent only different stages of the same basic process, there is practical clinical value in distinguishing between them. We therefore subscribe to the suggestion (4, 6) that the term "nephrocalcinosis" be reserved for that type or degree of diffuse calcification of the renal parenchyma that can be demonstrated roentgenographically, and that the non-specific designation "renal parenchymal calcification" be used to refer to that which is identifiable only by histologic examination.

SUMMARY AND CONCLUSIONS

Postmortem roentgenograms of kidneys from 210 cases of histologically identifiable calcification of the renal parenchyma were studied. An attempt was made to correlate the roentgenographic demonstrability of deposits of calcium with their histopathologic features, chemically determined quantity, and associated causative disorders. The following conclusions appear to be justified:

Parenchymal calcification commonly exists in kidneys to a degree, extent, or type that is not discernible roentgenographically.

In certain instances renal calcification which histologically is not readily distinguishable from that which cannot be demonstrated roentgenographically does cast characteristic and identifiable shadows on the roentgenogram.

The cases in this series in which we have been able to demonstrate the presence of calcium deposits roentgenologically were associated with the following causative disorders: primary hyperparathyroidism, chronic pyelonephritis, and chronic glomerulonephritis. This group of cases did not include examples of nephrocalcinosis due to hyperchloremic acidosis, recurrent renal lithiasis, or hypercalciuria of undetermined etiology, which comprise the only other causative disorders known to be responsible for producing roentgenologically demonstrable nephrocalcinosis.

The reasons why some renal parenchymal calcification is and some is not apparent roentgenographically are not clear and need further study. The quantitative chemical determinations in general indicate a higher content of calcium in those kidneys with roentgenologically demonstrable deposits. On the other hand, neither gross nor histologic estimation of degree of renal calcification is indicative of its roentgen demonstrability.

Roentgenography appears to be a practical method for separating a small group with a certain degree or type of calcification from the large group of cases with histologically demonstrable renal calcification. This small group has certain clinical implications that do not apply to other patients having renal calcification. The term "nephrocalcinosis" should be reserved for this type or degree of renal calcification which can be shown roentgenographically.

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SUMARIO

Descubrimiento Roentgenográfico de Calcificación Renal Identificable Histológicamente

En 210 casos que revelaban calcificación parenquimatosa identificable histológicamente, se tomaron radiografías renales después de la muerte. De ellos, 174 no mostraron signos roentgenológicos de calcificación, 30 mostraron depósitos locales de calcio, que, sin embargo, no podía considerarse que representaran la calcificación evidente histológicamente; sólo 6 (2.9 por ciento) revelaron típicos signos roentgenológicos de nefrocalcinosis.

Tratóse de correlacionar la demostrabilidad roentgenológica de la calcificación con las caracteristicas hístopatológicas, las determinaciones cuantitativas químicas del calcio y el estado etiológico.

Las determinaciones químicas cuantitativas indicaron en general un contenido mayor de calcio en los riñones que tenían depósitos observables roentgenológicamente. En cambio, el cálculo, ya macroscópico o histológico, del grado de la calcificación renal no resulta indicativo de la demostrabilidad roentgenológica de la misma. Encontráronse depósitos de calcio observables radiográficamente asociados con los siguientes trastornos etiológicos: hiperparatiroidismo primario, pielonefritis crónica y glomerulonefritis crónica.

Dedúcese que la radiografía parece ser un método práctico para separar un pequeño grupo de casos que tienen cierto grado o forma de calcificación del numeroso grupo que tiene calcificación observable histológicamente. Ese pequeño grupo posee ciertas connotaciones clinicas que no son aplicables a los otros casos. El término de nefrocalcinosis debe limitarse a la forma de calcificación renal observable roentgenológicamente.

Vesical Physiology Demonstrated by Cinéradiography and Serial Roentgenography

Preliminary Report[1]

FRANK HINMAN, JR., M.D., GERALD M. MILLER, M.D., ELDON NICKEL, M.D., and EARL R. MILLER, M.D.

ON APPLYING NEW technics to the study of an old problem, we have been forced to question some of the current concepts of the mechanism of urination, concepts which are based on cystometrographic studies (1–4) or on direct film or fluoroscopic observation (5–8). We were not able to convince ourselves that what seemed apparent on the fluoroscopic screen was not the result of our inability to separate rapid sequential movements, although these technics might profitably be explored by others.

With suggestions from Dr. Howard L. Steinbach, we made use of cinéradiography and serial roentgenography to produce sequences of cystograms during voiding. With the x-ray movies we were able to make exposures at the rate of 3.25 frames per second; with the Fairchild roll-film cassette, the time between films was 1.1 seconds. In this way we gained the dimension of motion without sacrificing the ability to study each film of the sequence individually and in relation to the rest of the exposures.

In the preliminary study described here, we limited ourselves to analyzing micturition in the normal female subject.

MATERIALS AND METHODS

Two hundred cubic centimeters of 7 per cent sodium iodide and 25 per cent Skiodan (or of 25 per cent Skiodan alone) was instilled by catheter into the bladder in normal nulliparous women. After the subject was placed before the radiographic apparatus, she was told to call out when she was about to void. At the call, the camera or the roll-film cassette was started. Some patients were studied during the entire time of emptying the bladder. Others were instructed to start and stop urination.

Radiographic Technic for the Fairchild Roll-Film Cassette: The patient was placed in the erect position facing the Fairchild roll-film cassette. Films were exposed for 0.6 second with intervals of 0.5 second between exposures. The tube-film distance was 36 inches. Other exposure factors were: 66 kv., 100 ma., 2 mm. aluminum filter, and a 10 × 12-inch field. The skin dose was 1.5 r per exposure. The total amount received by the patient was 25 to 30 r depending upon the duration of the study. This is about the amount received during four to five minutes of fluoroscopy.

Technic for Cinéradiography: The patient was placed in the erect position facing the 10 × 12-inch fluoroscopic screen. The exposure factors were: 110 kv., 100 ma., 0.25 mm. copper filter, 0.3 mm. copper h.v.l., 32 inches distance, 3.25 frames per second, total exposure 14 seconds. The camera was set at 4 feet from the screen, and the lens was set at *f* 0.85. The total skin dose was 50 r.

INTERPRETATION

We will not at this writing postulate what sequence of activities constitutes "normal micturition." Our study is still incomplete and, besides, normal voiding will vary from time to time depending on the subject's urge to void. If she has repressed the desire to void, she will then urinate in a different fashion than if she voids on command. Interpretations of

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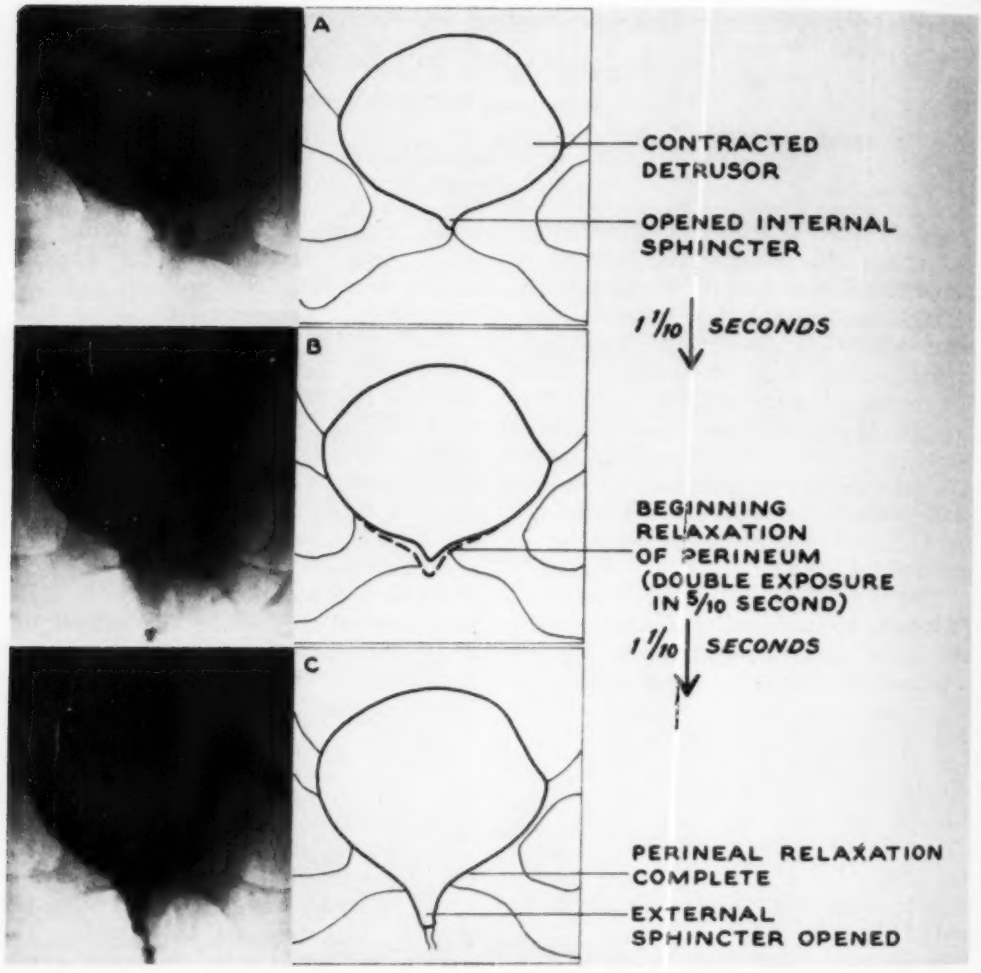

Fig. 1. Serial films of the bladder during initiation of urination in a normal nulliparous woman who already had the urge to void.

A. The detrusor is already contracted (spherical bladder outline) and the internal sphincter open (V-projection at bladder base).
B. On the command to void, the bladder base falls (seen as a blurred exposure).
C. The external sphincter (distal urethra) opens and urinary flow occurs.

bladder changes on voiding have been subject to error because the "state of the bladder" varies from situation to situation. At one extreme is the bladder in a resting state, sending few sensory stimuli through the spinal reflex arcs or to consciousness. At the other, is the painful bladder with overstretched muscle, sending so many stimuli that great difficulty is experienced in refraining from voiding. Between these extremes is the bladder producing enough sensory impulses to the cortex to cause a normal desire or urge to void. Naturally, the degree of relaxation or contraction of the bladder muscle, bladder neck, and urethral and perineal musculature will be different in each of these states. If we have, in addition, neurologic or anatomic abnormalities, the patterns taken by the elements of voiding can be quite varied. For each sequence, therefore, it is of great importance first to define "the state of the

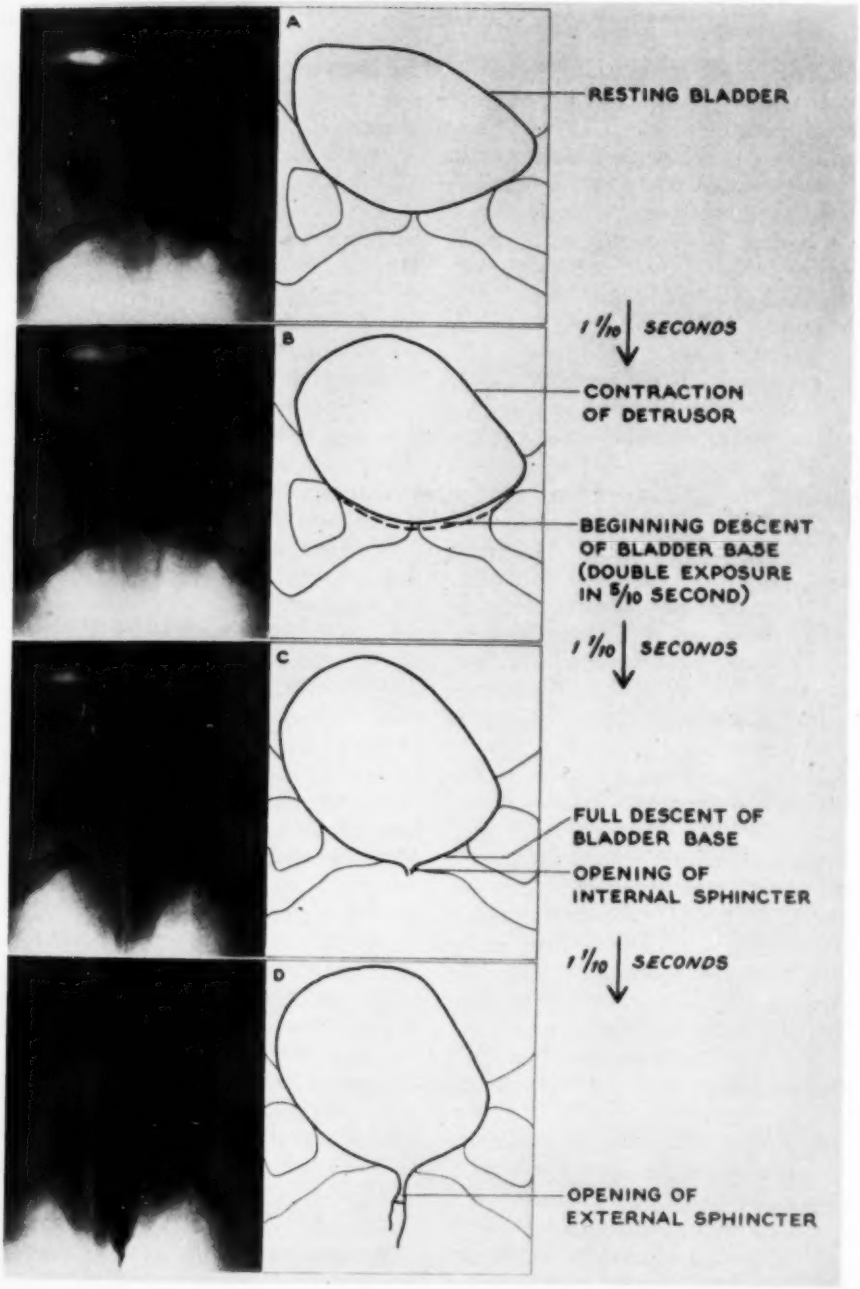

Fig. 2. Serial films during initiation of voiding in a normal nulliparous woman who had no urge to urinate but voided on command.

A. Normal resting bladder (flattened dome). The woman is told to start voiding.
B. Some detrusor contraction has occurred in the interval since the first exposure; yet the fall of the bladder base is seen during the period of exposure of this second film.
C. The internal sphincter opens.
D. The external sphincter opens.

bladder" at the moment of study, in order to interpret the physical pattern of pressure and form seen on the films.

As used here, detrusor contraction will mean the contraction of the entire bladder wall, including the trigone. The internal sphincter is that portion of the detrusor which encircles the inner end of the urethra. The external sphincter, under voluntary control, surrounds a segment of the distal two-thirds of the urethra.

It appears that "normal voiding" is an integrated activity in which usually we see first detrusor contraction, with opening of the internal sphincter. Detrusor activity is followed by lowering of the bladder base by relaxation of the perineum, and finally by opening of the "external sphincter." We have observed, however, that these motions can occur independently. To illustrate this, Figure 1A shows the bladder of a woman who has a great desire to void. The detrusor is contracted and the internal sphincter is open. On command, the bladder base is lowered (Fig. 1B) and the remainder of the urethra is opened (Fig. 1C). In contrast, Figure 2A shows the resting bladder of a woman without a desire to void. On command, she first contracts the detrusor, then begins to lower the bladder base (Fig. 2B). This sequence is proved by the finding that the detrusor had already moved before the outline of the bladder base blurred during the 6/10 second exposure. Finally the internal (Fig. 2C) and external (Fig. 2D) sphincters are opened.

Similarly, the activities involved in stopping urination can be isolated phenomena. Figure 3A shows the bladder of a woman voiding and commanded to stop. Figure 3B, taken 1.1 seconds after the command to stop, shows the isolated closure of the distal urethra ("external sphincter"), without significant raising of the bladder base. After 7.7 seconds (Fig 3C), the base begins to rise, and finally the internal sphincter closes and the detrusor relaxes (Fig. 3D).

The additional use of cinéradiography has allowed integration of these activities and has been impressive in showing the "bouncing" quality of urination through descent of the bladder base (Fig. 4).

DISCUSSION

Muellner's (9–12) concepts disagreed with earlier interpretations of the mechanism of micturition. Part of the difference lies in the different states of the bladder leading up to voiding. By visually recording the rapid sequences, we have avoided some of the pitfalls inherent in fluoroscopic observation. We cannot here discuss all aspects of previous studies of vesical function, but will relate our studies to the most recent ones, those of Muellner.

Muellner states that increased abdominal pressure and relaxation of the pubococcygeus muscle (perineum) initiates the voiding reflex. We have seen rather that the detrusor may contract first, accompanied by opening of the internal sphincter, this being followed by descent of the bladder base. The detrusor may contract concomitantly with opening of the internal sphincter, long before the base descends and the external sphincter opens (Fig. 2B). Muellner also states that at no time did he "observe the urinary stream shut off first in the region of the external sphincter" (7, p. 236). We, however, have recorded the contrary (Fig. 3B). The internal sphincter and the detrusor appear to be reciprocally innervated—an older concept (3)—for this sphincter does not open with descent of the bladder base but has in our cases opened with detrusor contraction. The converse is equally true at the termination of voiding.

IMPLICATIONS

We now have a tool for directly recording the component movements of normal and abnormal micturition. The implications for normal urination are apparent from the present study. We plan to study the parous female patient with or without stress incontinence (before and after repair), the normal male subject, and the male patient with prostatic enlargement,

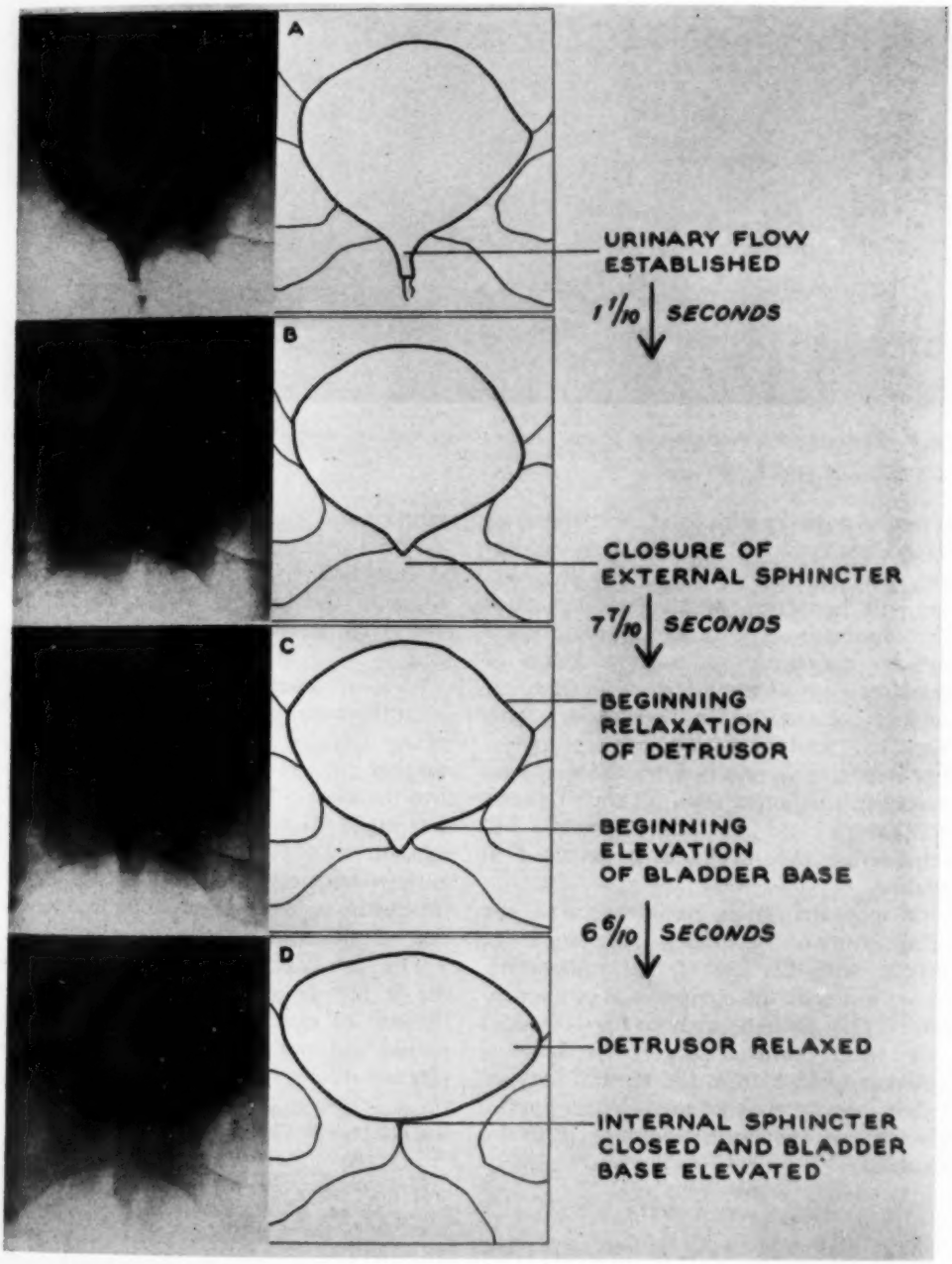

Fig. 3. Serial films of cessation of voiding on command in same female subject as Fig. 1.
A. The established stream. The patient is told to stop urination.
B. The external sphincter is closed as an isolated phenomenon (little or no associated elevation of the bladder base).
C. In film exposed 7.7 seconds later, the base starts to rise.
D. After 6.6 seconds more, the internal sphincter closes and the detrusor is relaxed.

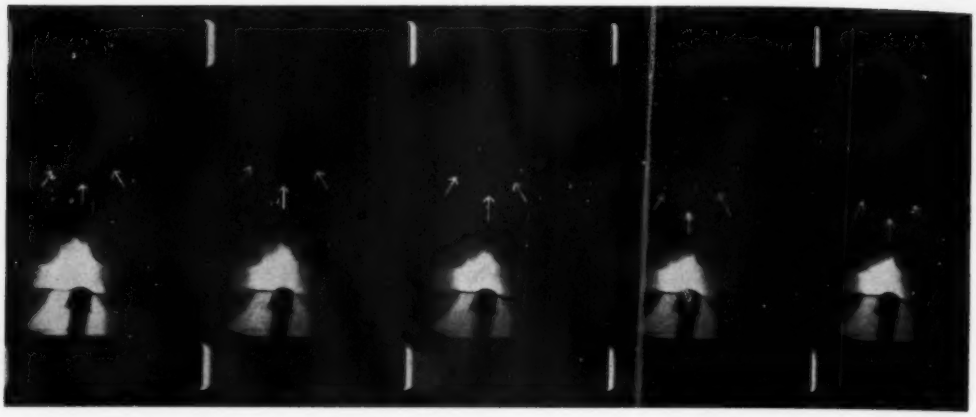

Fig. 4. Enlarged print of a segment of 16 mm. motion picture negative, made at 3.25 frames per second. Arrows show downward movement of bladder base.

as well as patients with fixed carcinomatous vesical necks and those who have undergone prostatectomy. Especially interesting will be study of the patient after abdominoperineal proctosigmoidostomy and of patients with various forms of neurogenic bladder. The alteration of voiding mechanisms in the bladder with large diverticula or with vesico-ureteral reflux needs to be studied by these special technics. Adjuncts such as simultaneous cystometry and electromyography will help define the mechanisms involved in voiding.

Of perhaps great importance to the future study of the abnormal bladder is the ability, with the use of cinéradiography, to separate out the components of micturition. The patient with a fixed bladder base from perineal surgery voids in a different fashion from the normal patient, but it may be that he merely uses part of the normal sequence, adapting it to his disability.

SUMMARY AND CONCLUSIONS

The Fairchild roll-film cassette and motion-picture fluoroscopy were used to obtain serial roentgenograms and cinéradiograms of the act of micturition in normal female subjects.

Urination, while co-ordinated, appears to be made up of several semi-isolated components, which may act independently. "Normal voiding" is a concept which must be qualified by a consideration of the "state of the bladder" when voiding begins—that is, in relation to the urge to void.

We have observed the following sequence in nulliparous female subjects: (1) isometric detrusor contraction and opening of internal sphincter; (2) relaxation of perineal muscles; (3) emptying contraction of detrusor; (4) isolated closure of external sphincter; (5) contraction of perineal muscles with elevation of base of bladder, (6) closure of internal sphincter and relaxation of detrusor.

The use of the technics described for the study of the normal and diseased bladder in the male and female patient should aid our understanding of vesical physiopathology.

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SUMARIO

La Fisiología Vesical Estudiada con la Cinerradiografía y con Radiografías en Serie: Informe Preliminar

Usando la cinerradiografía y la radiografía seriada para producir cistogramas sucesivos durante la micción en mujeres nulíparas normales, se demostró que la emisión de orina comprende varios componentes semiaislados que pueden obrar independientemente.

La "micción normal" es un concepto que debe ser limitado por una consideración del "estado de la vejiga" al comenzar el acto, es decir en relación con el impulso a orinar.

En las mujeres estudiadas, observóse la siguiente serie de acontecimientos: (1) contracción isométrica del detrusor y abertura del esfínter interno (la porción del detrusor que rodea el extremo interior de la uretra); (2) relajación de los músculos del perineo; (3) contracción evacuadora del detrusor; (4) contracción aislada del esfínter externo; (5) contracción de los músculos del perineo, con elevación de la base de la vejiga; (6) cierre del esfínter interno y relajación del detrusor.

Parece que el estudio de la vejiga normal y la patológica con las técnicas descritas debería ayudar a la comprensión de la fisiopatología vesical.

Postmortem Roentgenography with Particular Emphasis upon the Lung[1]

ROY R. GREENING, M.D., and EUGENE P. PENDERGRASS, M.D.

THE ROENTGEN examination of the chest is one of the most useful and widely employed tools in present-day medicine. It therefore becomes imperative that one understand its limitations as well as its positive values. Studies have been made of the subjective evaluation of chest roentgenograms (1, 5, 6) on the basis of a series of tuberculosis survey films interpreted by a group of experienced observers. Each member of the group made two or more interpretations of the roentgenograms independently at intervals of several months, and these separate interpretations (intra-individual) were compared. In addition, the interpretations of each individual in the group were compared with those of his colleagues (inter-individual). Not only did these studies show a considerable inter-individual variation in interpretation, but, more important, they revealed an intra-individual variation of 20 per cent.

Attempts at an objective evaluation of the examination itself have been made by Rigler, who states that a nodule 3 mm. in size can be detected in a technically good roentgenogram of the chest (17). This observation has been confirmed by Newell (15), at Stanford University, using physical objects of different sizes, shapes, and densities. Both of these studies were concerned with the small or minimal detectable lesion or object. Robbins (18), on the other hand, states that occasionally a lesion as large as 1 cm. in size may not be visible on the roentgenogram.

For years, we have been cognizant of the fact that the pathologists observe lesions in the excised lungs that we could not demonstrate upon antemortem roentgenograms. In 1944, therefore, we began to make a series of postmortem roentgenograms of the chest. These were then compared with the antemortem studies as well as with roentgenograms of the excised lungs. We were amazed to find not only that we were missing many small lesions but that in certain instances relatively large lesions were not seen, even in a restudy of the antemortem films. The scope of these studies has now been extended in a number of ways, including the radiography of operative specimens in which a similar experience has been encountered.

Descriptions of postmortem roentgen examinations have been published by others, initially by Allred and Garland (1) in 1935, later by Birkelo and Brosius (5) in 1938, and Beilin (3) in 1951. Our technic has improved with experience. The roentgenograms are made in the position employed for the living subject. The examination is made, therefore, in the erect posture. This is accomplished by suspending the body by means of an orthopedic traction apparatus with chin and occipital straps and buckles, connected to a metal cross bar with different slots for heads of different sizes. A wide canvas strap is placed beneath each axilla and connected to the side arms of the suspension bar. All belts are adjusted to the same length so that when the body is suspended in the erect position there is no unusual tension upon any one part. The center of the suspension bar is then connected to a heavy steel hook and the body elevated by means of a block and tackle, with the top of the pulley system anchored high on the wall or in the ceiling.

A simple wooden cassette holder is firmly attached to the wall. Just below

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[1] From the Department of Radiology, Hospital of the University of Pennsylvania. Accepted for publication in June 1953.

These studies are being continued under grants from the Penn Mutual Foundation for the Study of Malignant Disease and the Institutional Grant of the American Cancer Society and Atwater Kent Cancer Fund.

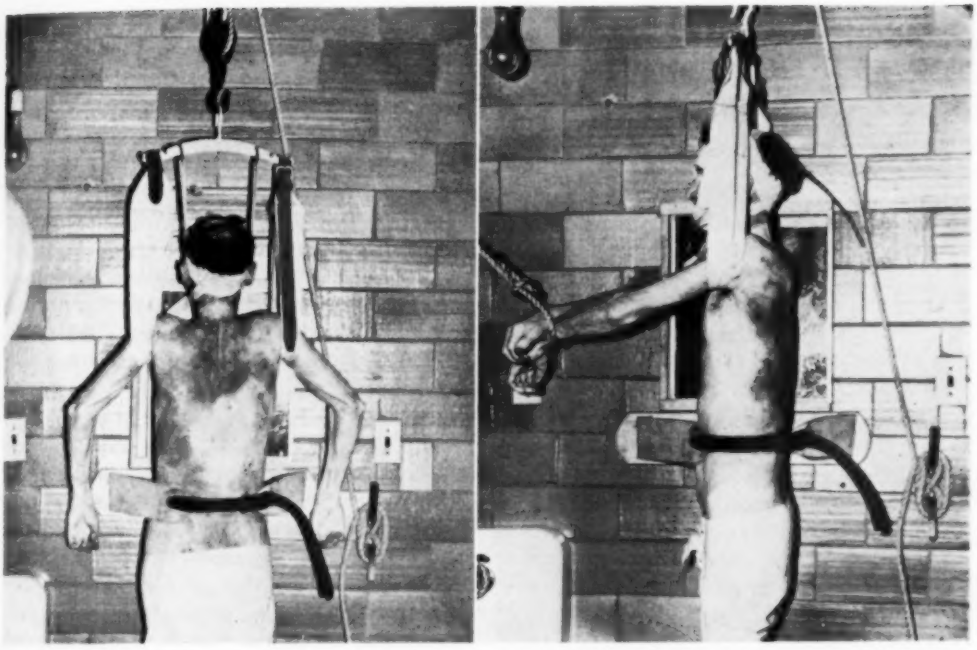

Fig. 1. Cadaver suspended in traction device for postero-anterior and lateral roentgenograms of the chest. See text for description of the suspension apparatus.

this another wide canvas belt is fastened to the wall. This is used to anchor the body firmly against the cassette holder (Fig. 1).

While the body is still on the litter, a No. 15 spinal needle is inserted into the trachea just below the cricoid, with the stilet in place. When the body is hoisted to the erect position, the weight of the abdominal contents is enough to suck air in through the spinal needle, from which the stilet has been removed, allowing good diaphragmatic excursion. With a little practice, we have found that it is possible, in the cadaver, to simulate the conditions under which inspiratory roentgenograms are made in a living subject. With the body suspended as described, roentgenograms are obtained in various conventional and unconventional projections (Fig. 2).

After the roentgenograms are made, the thorax is opened and the contents are removed by the Rokitansky method. If many pleural adhesions are present, an extrapleural dissection is carried out to prevent alveolar tearing and a resultant leak in the lung. The excess pleura can be carefully cut away after the lung, heart, and great vessels are removed. A large, heavy-walled rubber cannula is inserted into the trachea and tied in place. The outer end of the cannula is attached to an ordinary blood-pressure bulb or to a manometer, and the lung slowly re-inflated. With a little experience, one soon learns the amount of inflation necessary to simulate the living lung and not rupture the alveoli. Roentgenograms are made with the heart and great vessels attached and removed (Figs. 2 and 3). Cardboard holders or cassettes are used. Occasionally, in order to demonstrate very finely calcified lesions, the screen cassette holder with or without the Potter-Bucky diaphragm is of definite value. Almost any kind of x-ray generator may be used as the source of the x-rays, since the time of the exposure and the problem of motion are not modifying factors. It is important, however, we believe, to use a fine-focus tube.

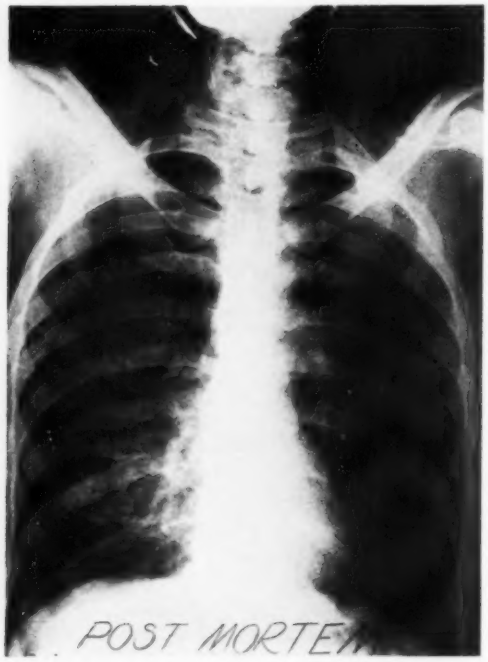

Fig. 2. Postero-anterior roentgenogram of the chest made after death. The patient has a carcinoma of the left upper lobe which showed as a shadow in the medial portion of the lung just above the hilus. This was much more apparent upon the antemortem roentgenograms.

Since we have employed this procedure, correlations have been made between the findings in the antemortem, postmortem, and excised-tissue roentgenograms and those observed, grossly and microscopically, in the tissue in over 300 cadavers. The results have been extremely informative. A similar attempt at correlation of the pathological and roentgenolgic findings has been made on surgical specimens but, because of the frequency of rupture or morcellation of the lung at the operating table, the results from this study have been less rewarding.

COMMENT

Massive pulmonary edema, cardiac enlargements, lobar and lobular consolidations in various areas of the lung, pleural and interlobar effusions, as well as all varieties of atelectasis, have been readily demonstrated. Our experience with these conditions is similar to that reported by others (3, 5). Our discussion will therefore be limited to intrathoracic masses and nodules. No effort will be made to comment on embolic lesions or lesions that

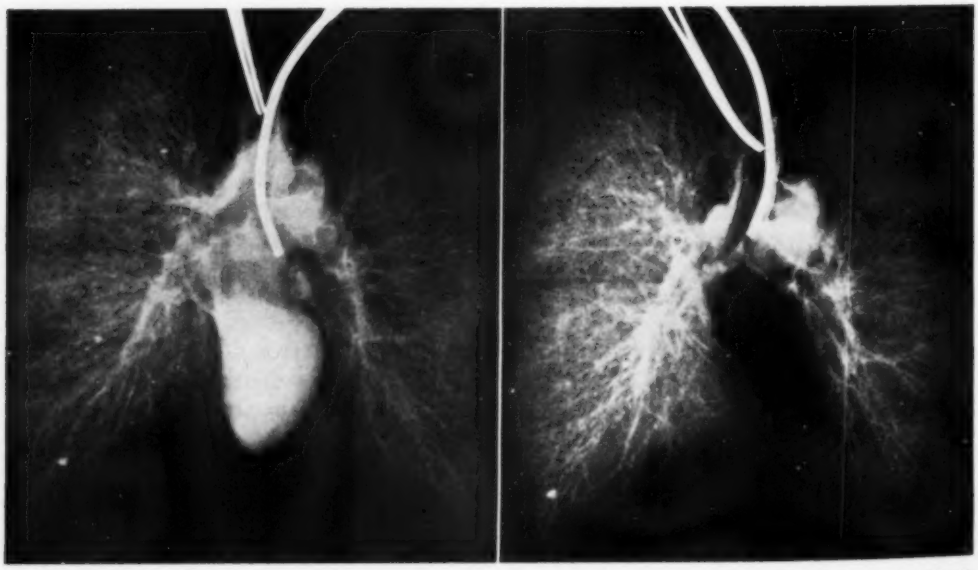

Fig. 3. A. Postero-anterior roentgenogram of the excised re-inflated lungs of the patient shown in Fig. 2. The heart and aorta are intact, and the tumor is visible in the lung above the left hilar structures.
B. The heart and aorta have been removed, allowing one to see the tumor to better advantage. A small metastatic lymph node previously unsuspected is now visible above the right main stem bronchus at its origin.

require careful dynamic studies (2, 7, 8, 12) for antemortem recognition.

The lesion most commonly escaping demonstration on the routine conventional roentgen examination of the chest is the small calcified or uncalcified nodule 3 mm. or slightly more in diameter. Particularly is this true of pleural and subpleural nodules along the lateral chest wall and over the domes of the diaphragm. Similar observations have been made at operation; that is, pleural lesions are seen and subpleural nodules are felt, which were not demonstrable on preoperative roentgenograms. Such a lesion was found to be present in nearly every postmortem lung examined. Frequently the shadows overlie the shadow of ribs and are not detected in the conventional study but are recognized on restudy.

The next most common lesion that fails to be recorded is found in patients who have obvious metastatic disease with a few visible nodules in each lung. Some of these may reach a diameter of 1.0 cm. In nearly every instance the inflated excised lung revealed many, many more nodules than had been suspected or could be counted in the antemortem study.

A third, although less common, type of lesion that is not demonstrated adequately is associated with some form of pneumoconiosis or lymphatic spread of cancer throughout the lung. Repeatedly, we have been impressed by patients with a positive history of considerable dust (silica) exposure who show no roentgen evidence of silicosis. Postmortem roentgenograms of the excised lungs in such cases have revealed diffuse nodulation, which has also been seen by the pathologist in his microscopic sections. In this type of case it often is quite difficult to obtain satisfactory studies because of heavy pleural adhesions resulting in alveolar tearing during removal of the lungs from the thorax, making their reinflation impossible. These observations seem to be good clinical proof of the concept, presented by Resink (16) and Newell (15), of a series of subliminal shadows, each in itself below the threshold of human vision, but when added together eventually reaching a size large enough to be appreciated by the retina. In principle, this same story is true of fine thread-like lymphatic metastases throughout the lung (13). Several patients with diffuse miliary tuberculosis also fall into this grouping. Their lesions were first found at postmortem examination.

Lesions in the mediastinum and close to the hili are commonly missed on conventional roentgenograms. They will not be discussed in this presentation because everyone is aware of the difficulty of diagnosis of involvement of the mid-line structures.

The rarest group of lesions failing to be recorded on conventional roentgenograms, but certainly the most spectacular, consists of solid nodules ranging from 1.0 to 5.0 cm. in diameter. Some of these have been completely undetectable in conventional, technically good roentgenograms of the living patient and likewise in the postmortem roentgenograms of the chest with all the organs *in situ*. They have been detected only on palpation by the pathologist and demonstrated only in roentgenograms of the excised reinflated lung. In the series of 300 patients examined, this type of lesion was discovered in 4. In 3 of the 4 patients an antemortem diagnosis of carcinoma of the lung had been made, but on evidence other than the presence of a mass. In one instance a small metastatic hilar lymph node led to the diagnosis of bronchogenic carcinoma. In this case the primary tumor mass measured 5 cm. in diameter. In a second patient the diagnosis was based upon a diffuse anaplastic extension of the tumor into the interstitial portion of the lung. The major mass, completely invisible, was 2 cm. in diameter. In a third patient, a small destructive lesion in a rib led to suspicion of a bronchogenic carcinoma. The mass in the lung, not connected with the rib, measured 2 cm. The fourth patient had a completely unsuspected primary carcinoma of the lung 2 cm. in diameter.

There are several explanations for inability to see certain lesions such as diffuse silicosis, widespread miliary tuberculosis, and certain types of metastatic cancer. These include completely subliminal shadows and those below the threshold of visibility (15, 16). Many undetectable lesions, large and small, occur in one of the relatively blind areas of the chest. These areas are along the parietal pleura, close to the ribs, and in the mid-line, where the shadow of the aorta, heart, and spine are very dense. A lesion in the peripheral portion of the apex of the lung is sometimes hidden. In each instance, as suspected by Robbins (18) we have found these areas to be so situated that it would be impossible to project a lesion upon well aerated pulmonary tissues without the confusion of overlying ribs, vertebrae, or major blood vessels. We feel, for this reason, that these lesions will continue to remain invisible in conventional roentgenograms of the chest made with present technics.

It is our belief that conventional roentgenography, although good, leaves much to be desired. For that reason, an investigation of the very high-voltage ranges has been initiated for use in diagnostic radiology.

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SUMARIO

La Radiografía Postmórtem con Atención en Particular al Pulmón

En 300 cadáveres, se tomaron radiografías del tórax, comparándolas con las tomadas antes de la muerte en los mismos casos, así como con las radiografías de los pulmones excindidos y reinflados, correlacionándose las observaciones con los hallazgos histopatológicos, obtenidos macro y microscópicamente.

Observóse que las lesiones que más a menudo evaden descubrimiento en las radiografías convencionales corrientes son los pequeños nódulos, ya calcificados o no, sobre todo pleurales o subpleurales, de unos 3 mm. de diámetro. Otras lesiones que no se descubrieron antes de la muerte fueron nódulos metastáticos múltiples de

cáncer, nodulación difusa en algunos casos de silicosis, metástasis filiformes esparcidas por todo el pulmón, las lesiones de la granulia y, más raramente, los nódulos carcinomatosos macizos de 1.0 a 5.0 cm. de diámetro.

La inobservación de esas lesiones tal vez se deba a su pequeño tamaño (inferior al umbral de visibilidad), su localización en zonas "ciegas" del tórax y la interposición de tejidos sobreyacentes. Opinan los AA. que esas lesiones continuarán siendo invisibles en la radiografía corriente del tórax, a la luz de lo cual han iniciado una investigación de los voltajes más altos para la roentgenografías de diagnóstico.

Torsion of the Lung Following Thoracic Trauma

A Case Report[1]

E. H. STRATEMEIER, M.D., and J. W. BARRY, M.D.

NUMEROUS CASES of thoracic and pulmonary trauma are reported in the literature. Descriptions are to be found of pneumothorax, hemohydrothorax, atelectasis, pulmonary edema, interstitial and alveolar hemorrhage, ruptured alveoli, and ruptured bronchi.

The following case is believed to be unique.

On April 29, 1952, at 5:55 P.M., a six-year-old white boy was admitted to the Pediatric Ward from the Emergency Room. He had been struck by a vehicle approximately thirty minutes before admission, suffering a compound fracture of the right humerus and possible internal injuries. There were multiple abrasions, and a tire mark was evident over the left lower chest anteriorly. Roentgenograms of the right humerus, chest, and abdomen were immediately obtained. The fracture of the humerus was at the mid point of the shaft, comminuted and compounded. The flat film of the abdomen disclosed no unusual findings. The chest roentgenogram showed irregular clouding of the upper half of the right lung field with a peculiar striation pattern in the mid-portion of the left lung field. This striation extended from the hilus laterally and then swept upward in a curving fashion. Bilateral pneumothorax was present, most pronounced on the left. Several fractured ribs were seen on the left in the mid-portion of the chest (Fig. 1).

Speculation as to the cause of the markings in the lung fields suggested a combination of pneumothorax with areas of atelectasis, pulmonary edema, and perhaps some hemorrhage within the lung tissue. The peculiar course of the striations on the left was not adequately explained.

The child was taken to the operating suite after shock was controlled and 200 c.c. of blood given. The wound of the humerus was cleaned, and immobilization was effected. The patient returned from surgery at 11:30 P.M. with an endotracheal tube in place. Reddish colored froth came from the tracheal tube and respiration became irregular and labored. Death occurred at 12:40 A.M., some six hours and forty minutes after admission.

At autopsy the external physical findings were again noted. On section the abdomen was found to be normal except for retroperitoneal hemorrhage

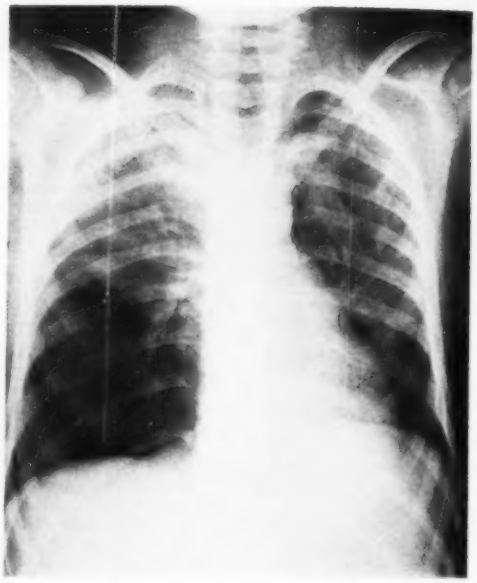

Fig. 1. Torsion of the lung due to thoracic injury

in the splenic area. The thoracic cavity showed the left lung to be rotated anteriorly, with its base assuming a superior position and the apex in the inferior position. The cavity contained 500 c.c. of frank blood. Interstitial hemorrhage was present. There was also a small amount of bloody fluid in the right pleural cavity. The heart was normal. Microscopic examination showed interstitial and alveolar hemorrhage.

Final Diagnosis: Automobile traumatism; torsion and inversion of the left lung; acute massive pulmonary infarction on the left, with hemothorax; pulmonary atelectasis and congestion on the right with interstitial pulmonary hemorrhage; perisplenic retroperitoneal hemorrhage; compound fracture of the right humerus; fracture of the fourth and fifth left ribs; multiple superficial abrasions, contusions, and lacerations; acute internal hemorrhage of the adrenals.

In view of the autopsy the sweeping striation seen in the left lung field is readily explained as the bronchovascular pattern of the lower lobe in the inverted position.

[1] From the General Hospital, Kansas City, Mo., E. H. Stratemeier, Radiologist; J. W. Barry, Assistant Radiologist. Accepted for publication in June 1953.

As a result of the pneumothorax, the upper lobe was collapsed (at least partially) at the time of roentgenography, and the striation in this direction was not readily apparent. It is assumed that sudden compression of the thorax by the weight of the vehicle as it passed over the lower chest caused collapse of the lower lobe and forced it cephalad. When the pressure was suddenly relieved, the lower lobe re-expanded in its new position, with the upper lobe depressed and only partially re-expanded.

A review of the literature revealed no similar example of thoracic injury. This case is reported with the hope that a serious traumatic thoracic condition may be more readily recognized.

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SUMARIO

Torsión del Pulmón Consecutiva a Traumatismo Torácico

Descríbese una secuela extraña de un traumatismo torácico en un niño de seis años. El enfermito había sido atropellado por un automóvil, que le había evidentemente pasado por encima de la porción inferior del tórax. Las radiografías revelaron un patrón peculiar de estriación en la porción media del campo inferior del pulmón izquierdo, extendiéndose las estrías desde el hilio hacia el lado y luego desplegándose hacia arriba en una forma curiosa. En la autopsia, se descubrió que el pulmón estaba rotado hacia el frente, tomando la base una posición superior y quedando el vértice abajo. Las estrías desplegadas representaban el patrón broncovascular del lóbulo inferior en su posición invertida.

A Homemade Inexpensive Manual Rapid Cassette Changer for Angiocardiography and Cerebral Angiography[1]

KENNETH W. TABER, M.D.

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THE PURPOSE of this article is to describe a manual rapid cassette changer which anyone can make inexpensively (at a cost of less than $60.00) and to mention two advantages which it offers over previous devices.

Briefly, the cassette changer consists of a "cookie sheet" to hold the cassettes as they are pushed manually through an elongated wooden channel which has lead covers on either side of the part of the body to be exposed. Wooden spacer blocks hold the cassettes in place in the "cookie sheet": a long narrow spacer at the top of the cassettes when they are placed in the tray with their longest dimension crosswise of the x-ray table (lengthwise of the channel), and a short block the width of the tray when their longest measurement is parallel to the table (crosswise of the channel). A Lysholm grid is placed between the lead covers to complete the upper surface of the channel.

Figure 1 shows the parts of the cassette changer with the technician holding the control rod in his right hand and the exposure switch in his left. Figure 2 shows the patient in place for a cerebral angiogram, with the needle in the internal carotid artery and the operator protected by the lower half of an old lead-rubber apron draped around the tube.

SPECIFICATIONS

The inside measurements of the wooden frame are $60\frac{1}{2} \times 13\frac{5}{8} \times \frac{5}{8}$ inches. The bottom of the channel is made of plywood and is nailed or screwed solidly to the sides. The inside surfaces of the sides are grooved near the top to accommodate the plywood and lead covers, each of which measure 24 $\times$ $14\frac{3}{8}$ inches. One plywood and lead cover is fixed at one end of the channel, while the other has stops in its grooves so that it may slide $1\frac{1}{2}$ inches from the near end of the channel. A $\frac{1}{4}$-inch hole (to accommodate a $\frac{1}{4}$-inch metal thumb key resembling a violin peg) is drilled through the end of one side of the channel at the proper place to bridge the groove for the movable lead cover so as to hold it $1\frac{1}{2}$ inches from the grooved end. One-inch squares are cut from $\frac{3}{8}$-inch plywood and nailed to the sides of the channel at points where they will guide the Lysholm grid and hold it in position over the opening in the middle of the channel. Four 4-inch wood clamps (C-clamps) suffice to hold the channel to the x-ray table.

The cassette tray, resembling a "cookie sheet," is made of galvanized tin. Its inside measurements are $39\frac{1}{2} \times 13\frac{1}{4}$ inches, with $\frac{1}{2}$-inch sides. A 1-inch square flattened metal cabinet handle is soldered to one end of this tray, fitting into the key-seat near the end of the solid metal control rod. The key-seat is a round hole through the control rod with a slightly smaller slot, opening the hole to one side of the rod so that the metal cabinet handle will be barely admitted when the rod is in a vertical position and will not slip out of the key-seat when the rod is in a horizontal position, as shown in Figures 1 and 2. The key-seat at one end of the rod must be 90° around the rod from the key-seat at the opposite end, so that the metal tits or stops to be used with one key-seat will not interfere with those to be used with the other. The control rod, then, is a solid metal rod $\frac{3}{8}$ inch in diameter and $31\frac{1}{2}$ inches long, not unlike a solid curtain rod. Approximately $\frac{3}{8}$ inch from each end is a key-seat. The metal stops for each key-seat are welded to the rod $7\frac{3}{8}$ and $18\frac{7}{8}$ inches respectively from one key-seat and on the

[1] Accepted for publication in May 1953.

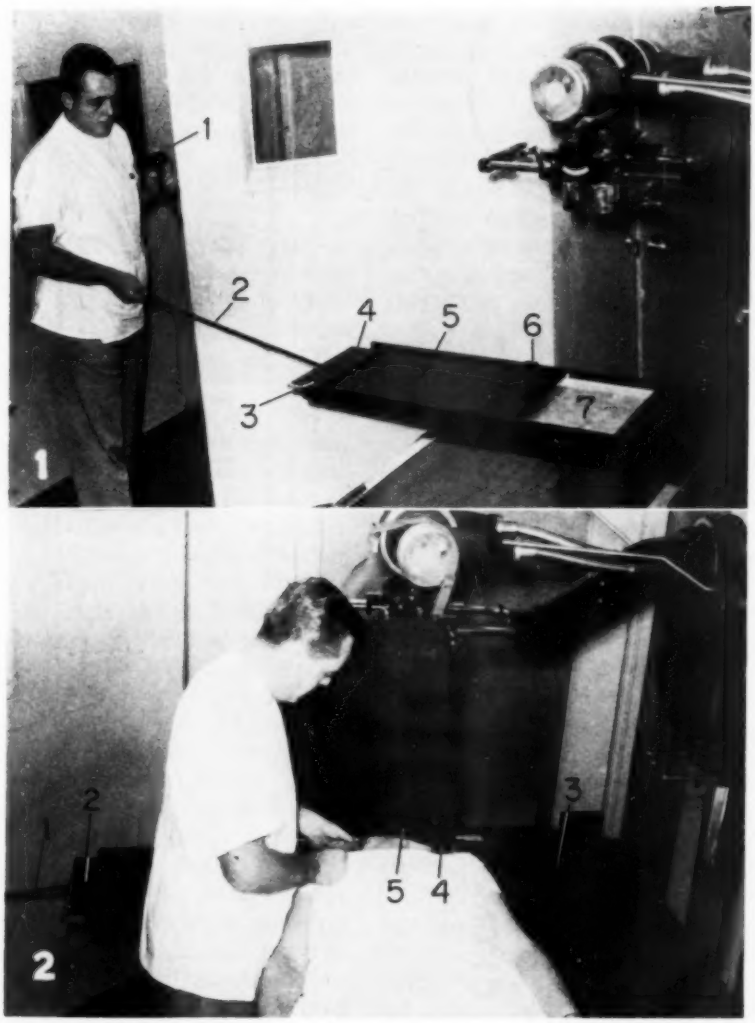

Fig. 1. Inexpensive homemade manual rapid cassette changer. (1) Exposure switch. (2) Control rod. (3) Thumb key. (4) Spacer block. (5) Movable lead cover. (6) One of the five guide blocks to hold the Lysholm grid in place. (7) Cassette tray ("cookie sheet") as seen through the exposure opening between the lead covers of the channel.

Fig. 2. Rapid cassette changer ready for operation. (1) Control rod. (2) Spacer block. (3) Stationary lead cover of the channel. (4) Lysholm grid under patient's head. (5) Lower half of leaded rubber apron to protect the operator, technician, and patient.

same quarter of the round as key seat No. 1. At 90° around the rod and on the same quarter of the round as the key-seat at the opposite end (key-seat No. 2) are welded two more metal stops $9^3/_4$ and $22^7/_8$ inches, respectively, from key-seat No. 2. A narrow marker strip is painted near key-seat No. 1, and a wide marker strip near key-seat No. 2, for quick reference as to which key-seat to engage in the metal cabinet handle for use with the cassettes across or lengthwise of the tray, respectively.

One plywood spacer measures $2^1/_8 \times 39^1/_4$ inches and is $^3/_8$ inch thick; the other is $5^3/_4 \times 13^1/_8 \times ^3/_8$ inches. A $^3/_4$-inch hole is drilled in the near end of each of these

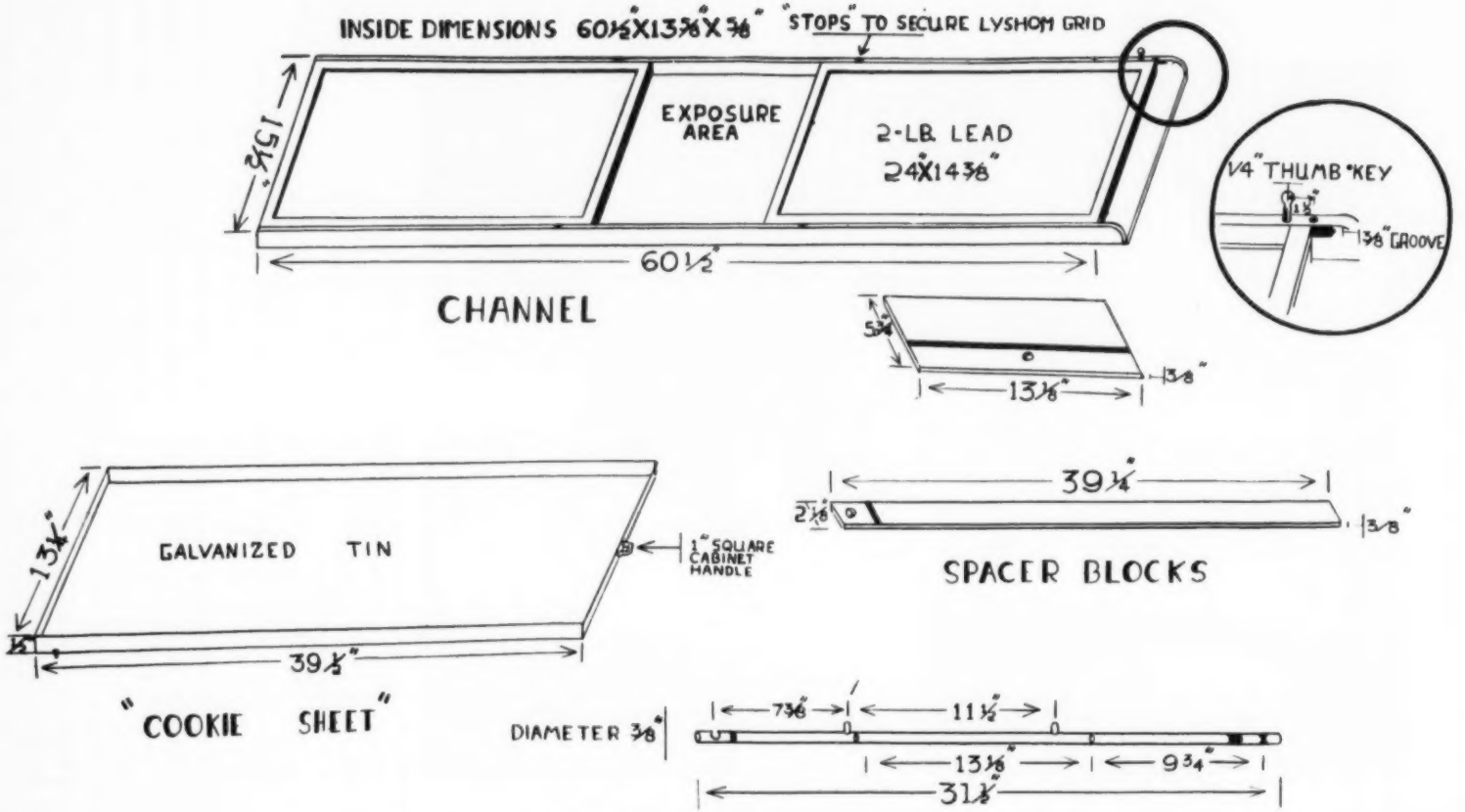

Fig. 3. Construction of the cassette changer.

spacers for convenience in removing them from the tray. On the same side as these finger holes are lines parallel to the x-ray table to indicate the depth that the tray is to be inserted into the channel for the first exposure.

OPERATION

The channel is clamped to the x-ray table and the Lysholm grid is placed over the opening in the channel. For angiocardiograms, a pillow is placed on the table above and below the channel for the comfort of the patient.

For angiocardiograms or anteroposterior cerebral angiograms, the three cassettes are placed with the 12-inch dimension crosswise of the "cookie sheet" and are jammed together at the end of the tray away from the technician. The wide stubby wooden block spacer is placed in the tray to finish filling it. The lead shield forming the top of the channel toward the technician is moved to its furthest position from the technician and held there by the metal thumb key in the hole for this purpose.

The metal control rod is held in a vertical position with the end bearing the key-seat on the same side of the rod as its nearest stops toward the technician, and with the narrow marker strip down. In this position, it is engaged in the metal cabinet handle at the technician's end of the tray. The control rod is then pivoted on this cabinet handle into the horizontal position (Figs. 1 and 2), and the tray is moved into the channel until the line on the spacer is below the near end of the lead cover.

The crystal is removed from a stop watch and a thin lead strip is pasted to its second hand. This is placed on the Lysholm grid over one corner of the cassette opening to indicate on the film, automatically, the time after the injection of the contrast medium at which each exposure was made. The patient is placed over the channel. The lead rubber apron is draped on the tube. At the designated time after the operator has injected the contrast medium, the first exposure is made.

As soon as the first exposure has been made, the technician depresses the control rod lightly, as he pushes it with the tray into the channel until the first metal stop on the control rod strikes the lower board of the channel to indicate that the second film is in place. After the desired interval, the second exposure is made. The procedure is repeated for the exposure of the third cassette, which is stopped in the proper location by the second metal tit on the same side of the metal control rod.

For cerebral angiography, the patient's head is then turned to the lateral position. The tray having been emptied, it is filled lengthwise by three 10 × 12-inch cassettes so placed that the 12-inch dimension is lengthwise of the tray (crosswise of the table). They are held in the tray by the long narrow wooden spacer, which likewise has a line across its near end parallel to the table to indicate the depth of insertion into the channel so that the first cassette will be in place under the patient's head. The movable lead shield forming the near half of the upper portion of the channel is moved $1^1/_2$ inches toward the technician after the thumb key has been removed to permit the extension of the opening under the patient's head. The control rod is moved into a vertical position and disengaged from the metal cabinet handle so that the rod may be turned end for end. The key-seat in the other end of the control rod is engaged in the metal cabinet handle on which it rotates to again bring the rod into a horizontal position, when the same procedure is followed to make the three exposures. Three exposures can be made within three seconds after the technician has had a little practice with "dry runs."

ADVANTAGES

The main advantages of this cassette changer over some of those previously described are found in its use for angiocardiograms.

First, the interval between the injection of the contrast medium and the exposures can be controlled by a stop watch to coin cide more nearly with the ether time an

Decholin time, as, for example, in the case of a mitral stenosis when the ether time may be normal but the Decholin time is greatly increased.

Second, the channel is sufficiently long to act as an armboard and operating table when the operator cuts down on a vein with the patient in position for an angiocardiogram.

SUMMARY

A simple manual rapid cassette changer, which can be made in the workshop at home, is described, with full specifications and details of operation. The advantages of this cassette changer in cardioangiography are indicated.

ACKNOWLEDGMENTS: Appreciation is hereby expressed to the Santa Ana Community Hospital for materials; to Cecil Miller, our engineer, for making this rapid cassette changer; to Ed Pearson, R.T., and Jim Pursell, R.T., for their ideas; to Mrs. Frances M. White for preparing the manuscript; and to Robert F. Dodson for the drawing reproduced in Figure 3.

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SUMARIO

Rápido Cambiachasis Manual, Poco Costoso y Hecho en Casa, para la Angiocardiografía y Angiografía Cerebral

La descripción de esta sencillo cambiachasis rápido y manual, que puede fabricarse en casa a poco costo, contiene estipulaciones completas y detalles del funcionamiento. La bandeja de chasis, parecida a una artesa llena de bollos, sostiene a los chasis al ser empujados a mano a través de una canal alargada de madera, provista de tapas de plomo a ambos lados de la parte del cuerpo que va a exponerse. Se usan cantos espaciadores de manera para sujetar en su sitio a los chasis. Para completar la cara superior de la canal, se coloca una rejilla de Lysholm entre las tapas de plomo. Para indicar en la película el intervalo en que se hace cada exposición, se coloca un reloj de segundos muertos, con una delgada tira de plomo pegada sobre la segunda manecilla, sobre la rejilla, en una esquina de la abertura del chasis.

Este accessorio ha resultado en particular útil para la angiocardiografía.

The Use of Conventional Depth-Dose Tables in Divided-Port X-Ray Therapy

Transmarginal Radiation and Depth-Dose Distribution[1]

GERHART S. SCHWARZ, M.D., and A. R. ISLAM, M.D.

DIVIDED-PORT therapy is the term employed whenever two or more small ports are substituted for one large port. It is understood to indicate that type of treatment in which a large skin area is covered by several small ports which may be contiguous or separated from each other by short distances, the x-ray beams entering the body through these portals being parallel or approximately parallel to each other. If the beams converge, the arrangement would have to be classified as cross-firing and would not fall within the scope of this paper. Cross-firing and divided-port therapy are often combined, *e.g.*, when each of two opposed body surfaces is treated through divided ports.

There are two types of divided-port therapy. One method consists of using a cone smaller than the total treated area, covering only one port at a time. With this technic, each of the smaller ports is covered by a separate beam, the target being aligned over the center of each individual port. The individual small ports are thus treated in succession. This method, since it requires that the x-ray tube be centered individually over each port while it receives its share of radiation, might, for the purpose of distinction, be called multicentric port division. The other method, which might be termed monocentric, consists in placing lead strips or a lead (rubber) grid in the path of a single x-ray beam, thereby dividing it into several smaller beams simultaneously.[2]

In the estimation of depth dosage in multicentric divided-port therapy, the contribution of scattered and penumbral radiation from neighboring beams is often overlooked. In consequence, doses are determined from depth-dose tables for the central axis of each beam separately as if it were not accompanied by any neighboring beam, and it is assumed that this is a fair representation of dosage throughout the area of the combined ports. While it is generally recognized that a point by point determination of depth doses by means of isodose charts is preferable, particularly if these include transmarginal radiation, this is frequently neglected. Many private radiologists and those working in smaller radiotherapy departments continue to rely exclusively on depth-dose tables because they do not have at their disposal (in addition to their regular x-ray technician) the services of a physicist or a technician trained to do the calculations. Even in those departments in which the use of isodose charts is routine, depth-dose tables are still employed as a rapid check on the tumor dose and as a means of planning tentatively the number and the spacing of treatments to be administered.

PURPOSE OF INVESTIGATION

The purposes of the investigation here reported were to find a simple means of adapting existing depth-dose tables to divided-port therapy by correcting for the transmarginal radiation which each x-ray beam produces and which has to be added to each neighboring beam, and to determine whether the error resulting from the neglect of this radiation is of a magnitude that justifies its correction in clinical radiotherapy.

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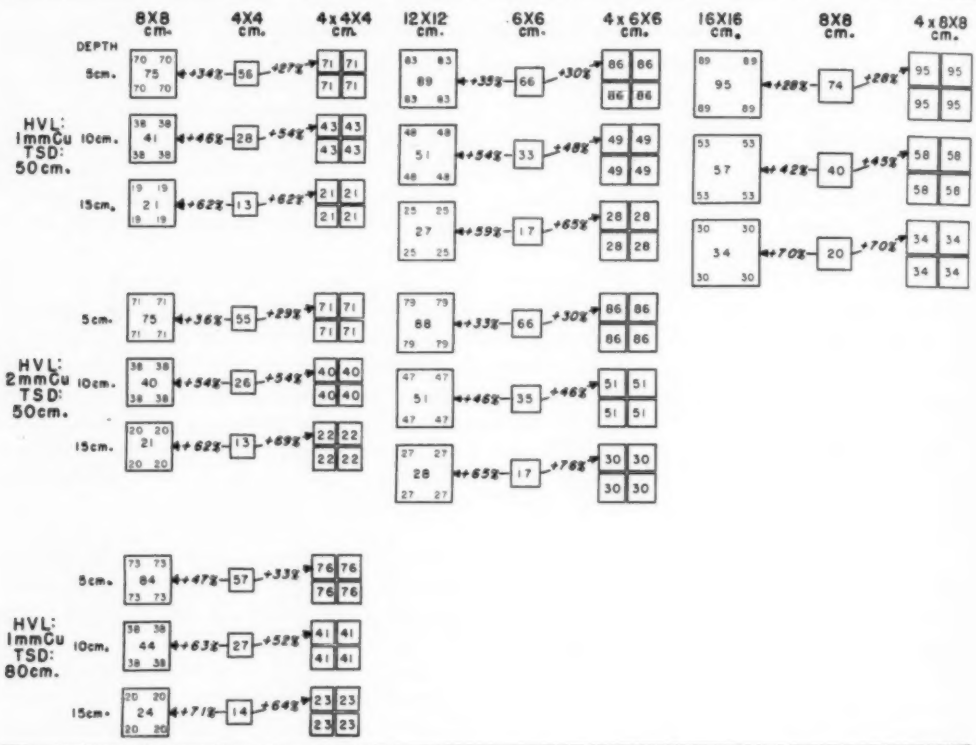
[1] From the Department of Radiology of the College of Physicians and Surgeons, Columbia University, and the Radiological Service of the Presbyterian Hospital, New York. Presented at the Thirty-ninth Annual Meeting of the Radiological Society of North America, Chicago, Ill., Dec. 13–18, 1953.

[2] The depth-dose distribution in monocentric port division has been published by others (5, 8, 10) for particular fields used in the treatment of carcinoma of the cervix and is not discussed here. Unpublished measurements for a greater variety of split fields also exist (9).

TABLE I: INFLUENCE OF PORT DIVISION ON DEPTH DOSE (r PER 100 r IN AIR). QUADRIPARTITION OF PORT SIZES 8 × 8 CM. TO 16 × 16 CM.



This transmarginal radiation consists partly of radiation scattered outside the direct beam and partly of penumbral radiation due to the large focal spot. The latter contribution may be reduced by careful diaphragming; the former is unavoidable.

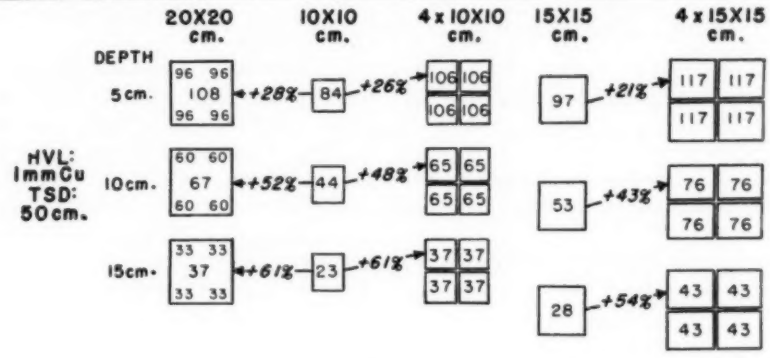
It is not always customary in multicentric divided-port therapy to include in the calculation this radiation reaching beyond the geometrical edge of the beam. Computing the depth dose, when irradiating a 20 × 20-cm. area by covering it with four 10 × 10-cm. ports, is then accomplished merely by looking up the depth dose listed in the table for a single 10 × 10-cm. port. No allowance is thus made for contribution of radiation by neighboring beams. Obviously, depth-dose tables list only the depth dose within the central ray of a single beam and cannot take into account combinations of several beams as they occur in divided-port therapy. Some isodose charts likewise lack a graphic record of transmarginal radiation.

APPLICATION OF DIVIDED PORT TECHNIC

The divided-port technic is applied to many deep-therapy cases today. The purpose of this procedure is sixfold:

(1) It is used when the area to be covered is larger than the largest cone available, the window of the x-ray tube being the limiting factor. This is particularly true in the treatment of generalized neoplasms, when the trunk of the patient is to be covered by four or six 15 × 15-cm. portals. This is necessary because the largest single skin area that can be covered without vignetting is for most x-ray machines 20 × 20 cm. at a target-skin distance of 50 cm.

TABLE II: INFLUENCE OF PORT DIVISION ON DEPTH DOSE (r PER 100 r IN AIR). QUADRIPARTITION OF 20 × 20-CM. PORT AND QUADRUPLICATION OF 15 × 15-CM. PORT.



(2) It is applied if small ports can be arranged in such a manner as to exempt a certain structure in the depth from irradiation, *e.g.*, two 10 × 15-cm. ports separated by an interspace to save the cervix from irradiation in a pelvic cycle.

(3) Conversely, it is employed in order to increase or accentuate the depth dose to certain structures over that obtained through a single large portal. Using two contiguous 10 × 15-cm. portals in lieu of a single 15 × 20-cm. port over the pelvis will produce a large increase of the depth dose to mid-line structures such as bladder or uterus in comparison to the dose delivered to more laterally located organs.

(4) It is found useful, also, when the purpose is to produce a more uniform depth-dose distribution at the margin of the treated area than is possible with single large cones. Substitution of four 10 × 10-cm. portals for one 20 × 20-cm. port (or four 15 × 15-cm. portals for a 30 × 30-cm. port) will avoid the undesirable dosage drop that exists at the edges of all larger beams.

(5) It is of benefit when one large cone cannot be fitted well to an irregularly shaped body surface.

(6) Finally, the divided-port technic serves to reduce the volume dose of each treatment while maintaining a tissue dose in each beam of a magnitude which could not be tolerated otherwise, if delivered in one session.

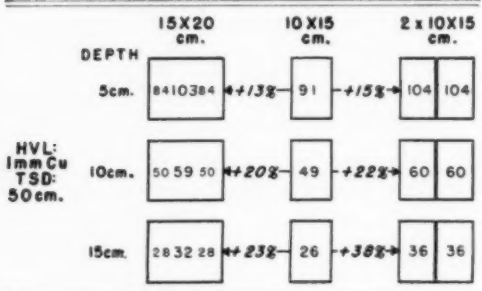
METHOD OF INVESTIGATION

Two qualities of radiation were used: 200 kv.p. with a h.v.l. of 1 mm. copper and 200 kv. p. with a h.v.l. of 2 mm. copper. Two target-skin distances were tested: 50 cm. and 80 cm. All measurements were made by embedding a 25-r (13 mm. in diameter) or 100-r (10 mm. in diameter) Victoreen condenser thimble chamber in various locations of a 30 × 30 × 30-cm. Masonite phantom and comparing the dose thus determined with the air dose measured with the same chamber in the central beam at 50 and 80 cm. from the target at a cross section of the beam of 15 × 15 cm.

RESULTS

Substitution of four 4 × 4-cm. ports for one 8 × 8-cm. port, four 6 × 6-cm. ports for one 12 × 12-cm. port, four 8 × 8-cm. ports for one 16 × 16-cm. port, and four 10 × 10-cm. ports for one 20 × 20-cm. port produced the depth-dose relationships shown in Tables I and II. The findings indicate that neglecting the transmarginal radiation produces an error of 27 to 33 per cent at a 5-cm. depth, of 45 to 54 per cent at a depth of 10 cm., and of 62 to 76 per cent at a depth of 15 cm. Somewhat smaller increases in depth dose are encountered when dealing with 15 × 15-cm. portals (Table II). This is explained by the fact that a combination of two or four

TABLE III: INFLUENCE OF PORT DIVISION ON DEPTH DOSE (r PER 100 r IN AIR). BIPARTITION OF 15 × 20-CM. PORT.



portals of this size extended to the edge of the phantom, the largest dimension of which was 30 cm. This reduced somewhat the amount of scattered radiation which a beam could contribute to its neighbors. It is felt, however, that the same condition prevails also in clinical radiotherapy, since covering the patient's trunk with two or four 15 × 15-cm. portals will bring the edge of the cone to or beyond the lateral margins of the body. Moreover, the difference is small enough to neglect it in clinical calculations.

In both series all measurements were confined to areas where there was no geometrical overlap of beams. Since all x-ray beams used in conventional therapy are divergent, there is always an overlap in the depth of any contiguous port combination. This overlap is confined to a narrow central zone in the depth of the phantom and is distinct from the transmarginal contribution.

As shown in Tables I and II, the error produced by neglect of transmarginal radiation is in rough approximation independent of the total area covered by the four ports. Transmarginal radiation apparently always increases the depth dose in the central ray of each small beam so that it about equals the depth dose in the central ray of the original large beam for which the small beams have been substituted. This makes possible an extremely simple correction applicable to conventional depth-dose tables for any listed depth. All the user has to do is to determine from the table the depth dose for that port which equals in size the sum of all small contiguous ports (expressed in square centimeters).

If this sum exceeds 400 sq. cm., which is usually the largest area listed in conventional depth-dose tables, one can extrapolate. Such an extrapolation is limited, because with very large ports less transmarginal radiation reaches the central axis of neighboring beams. Our tests have shown, however, that it can be extended up to a total area of 900 sq. cm. (four 15 × 15-cm. ports) at a permissible loss of accuracy in the following manner:

Example: A depth-dose table for a certain quality of radiation (h.v.l. 1.0 mm. Cu; T.S.D. 50 cm.) may list the following doses per 100 r in air for a depth of 7 cm.: 100 sq. cm., 63 r; 225 sq. cm., 76 r; 400 sq. cm., 85 r. This indicates that at this particular depth, quadrupling the area (from 100 to 400 sq. cm.) will increase the dose by 35 per cent. This will hold true, also—in approximation—if one quadruples the 225-sq. cm. port (15 × 15 cm.). The dose for this port, namely 76 r, must therefore likewise be increased by 35 per cent if the area is to be quadrupled. The result reads 102 r for an area covered by four 15 × 15-cm. ports (= 900 sq. cm.).

One is thus able to determine this dose, although the table listed no area larger than 400 sq. cm.

As is shown in Tables I and II, there is also a slight though noticeable gain in depth dose if one compares the depth dose in the quadrant of the large beam with that in the center of the small beam. In the case of a single large beam, its quadrants receive oblique marginal rays, whereas in the case of four small beams each quadrant of the same area covered receives a central beam. This probably explains the superiority of the depth dose distribution if several small beams are substituted for one large beam.

The combination of two contiguous 10 × 15-cm. portals was compared with a single 15 × 20 cm. portal (Table III). Here, also, the same relationship exists between a single small port, two small ports, and a single large port. For this particular combination, the error resulting from the neglect of transmarginal radiation is 15 per

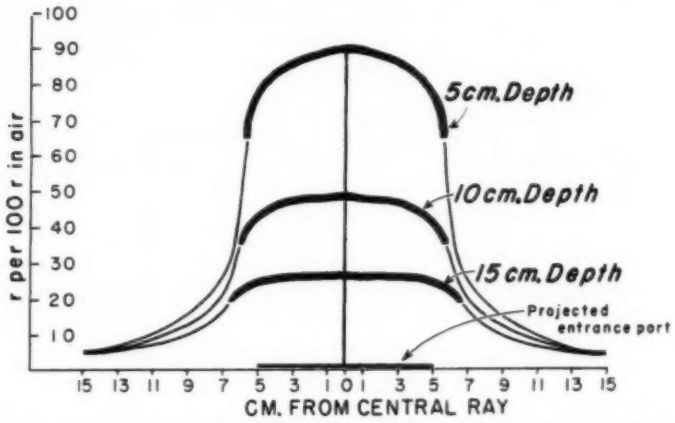


Fig. 1. Dose distribution along the short axis of a beam entering the phantom through a 10 × 15-cm. portal and its transmarginal extension. Heavy lines indicate intramarginal radiation, *i.e.*, radiation within the geometrical confines of the x-ray beam. Thin lines indicate transmarginal radiation. h.v.l., 1.0 mm. Cu; T.S.D., 50 cm.

cent at a depth of 5 cm., 22 per cent at a depth of 10 cm., and 38 per cent at a depth of 15 cm. Again the error can be corrected in approximation if one simply calculates for the larger area representing the sum of the two smaller ports.

The dose distribution across the short axis of a beam entering the phantom through a 10 × 15-cm. port was then measured. Measurements were extended beyond the geometrical margin of the beam as far as the size of the phantom would permit (15 cm. to either side of the central ray). The findings are shown in graph form in Figure 1. At all three levels (5, 10, and 15 cm. depth) the transmarginal radiation is considerable. The dose distribution across the short axes at a depth of 10 cm., if two contiguous 10 × 15-cm. portals are used, is shown graphically in Figure 2a. The increase over the dose within a single beam is significant. The narrow zone of overlap measuring 2 cm. in width, which is caused by the divergence of each beam (at a depth of 10 cm. each beam has a cross section of 12 × 18 cm. *vs.* 10 × 15 cm. at the entrance port), shows, of course, a much greater increase, but this should be disregarded in the consideration of the problem of transmarginal radiation.

If one separates the two 10 × 15-cm. entrance ports from each other by 3 cm., no overlap occurs at any depth less than 15 cm. At a depth of 10 cm. the two

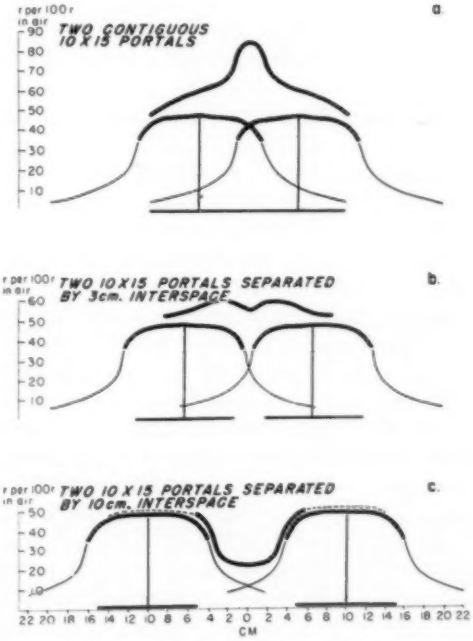


Fig. 2. Ten-centimeter depth-dose distribution along the long axis of a combination of two 10 × 15-cm. ports as function of port separation. Solid lines indicate measured values; dotted lines, estimated values; heavy solid lines, intramarginal radiation and/or total dose of combined intramarginal and transmarginal radiation; thin solid lines, transmarginal radiation.

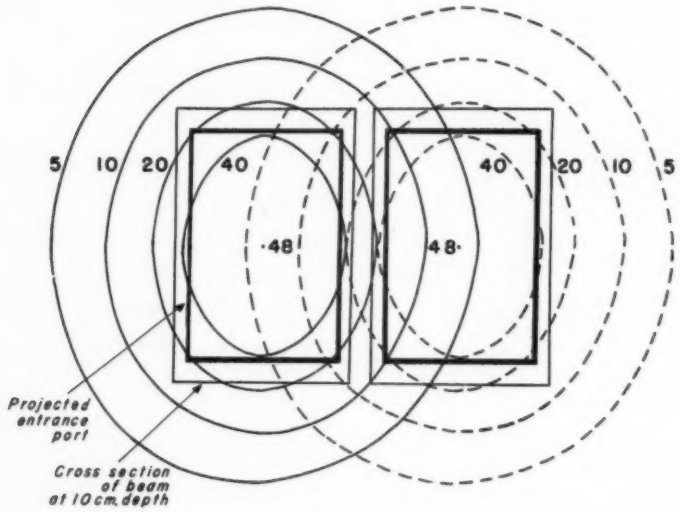


Fig. 3. Transverse cross section through same arrangement as shown in Fig. 2b. Isodose curves of each individual beam at a depth of 10 cm. show contribution of one beam to the other. Transmarginal isodose curves of one beam intersect intramarginal curves of the other one and *vice versa*.

beams are still separated from each other by 1 cm. The resulting dose distribution at this depth is shown graphically in Fig. 2b. The transmarginal radiation is still considerable, so that even in the interspace between the two beams the dose is higher than the highest dose produced by direct radiation in the center of a single beam. Of interest also is the fact that this degree of port separation produces the most uniform depth-dose distribution. It seems as if the interspace between the two beams were completely wiped out by the transmarginal radiation when the entrance ports are separated by a distance of 3 cm.

A transverse cross section through the same beam combination at a depth of 10 cm. is shown in Figure 3. It contains isodose curves for each individual beam, demonstrating the interaction of one upon the other.

Since this is a frequently used combination, particularly in the treatment of pelvic lesions, the influence of distance of separation upon the depth dose has been investigated. It was found that the dose in the central axis of each 10 × 15-cm. beam varied with the distance of separation as shown in Table IV. There is thus still an increase of the depth dose within each beam over that found in a single unaccompanied beam, even at a separation of 5 cm.

The dose within the interspace between the two beams can be calculated from such curves as those shown in Figure 1. These

TABLE IV: INFLUENCE OF SEPARATION UPON THE DEPTH DOSE IN THE CENTRAL AXIS OF TWO ADJACENT 10 × 15-CM. BEAMS

(Figures indicate r per 100 r in air to each of the two ports. H.v.l.: 1 mm. Cu; T.S.D.: 50 cm.)

Distance of Separation →	0 cm.	1 cm.	2 cm.	3 cm.	4 cm.	5 cm.	Undivided Large 15 × 20 cm. Port*	Single Unaccompanied 10 × 15-cm. Port
5 cm. depth	104	102	99	98	97	96	84	91
10 cm. depth	60	58	57	55	54	53	50	49
15 cm. depth	36	34	33	32	31	31	28	26

* Measured below the center point of each half of the field. See Table III.

TABLE V: INFLUENCE OF SEPARATION BETWEEN TWO 10 × 15-CM. ENTRANCE PORTS UPON THE DEPTH DOSE WITHIN THE INTERSPACE BETWEEN THE TWO BEAMS

(Figures indicate r per 100 r in air to each of the two ports. H.v.l.: 1 mm. Cu; T.S.D.: 50 cm.)

Distance of Separation of Ports →	0 cm.	1 cm.	2 cm.	3 cm.	4 cm.	5 cm.	6 cm.	7 cm.	8 cm.	9 cm.	10 cm.
5 cm. depth	154	130	85	64	51	45	39	35	31	29	26
10 cm. depth	86	77	70	56	41	37	33	30	28	25	23
15 cm. depth	48	46	43	38	33	29	25	23	21	19	18

calculations were made for the exact midpoint of the interspace at depths of 5, 10, and 15 cm. and found to be as shown in Table V.

In Table V all figures pertaining to a port separation of 3 cm. or larger represent interspace doses produced solely by transmarginal radiation (overlapping of beams being absent). At a depth of 10 cm., for example, the dose in the mid-line between the two separated beams measures 37 per cent of the air dose delivered to each port if the ports are separated from each other by 5 cm.

The dose distribution for a depth of 10 cm. is shown in Figure 2c and in Table V if the two ports are separated from each other by 10 cm. It indicates that it is virtually impossible to prevent some radiation from reaching the mid-axis of the pelvis when treating the parametria (23 per cent of air dose with 10 cm. separation).

DISCUSSION

The measurements here presented demonstrate the important role of the transmarginal radiation upon the total depth dose in conventional divided-port therapy. Radiologists should be cognizant of this and take it into account when estimating depth doses from tables. If not, large errors may be introduced. If contiguous or adjacent ports are to be employed, depth doses should be obtained from the table for the area which is the sum of all these.[3] It must be clear, however, that the air doses for all small beams are not to be added. For example, if 150 r in air are administered to each of four contiguous 10 × 10-cm. fields, the approximate depth dose within the region will be found from a table, for a 20 × 20-cm. field, *for 150 r in air*, not for 600. If two of the small fields are treated each day, the corresponding large field dose is delivered only every other day.

In this connection, the question may be raised as to whether the biological effect is the same if half a field is irradiated every other day as if the whole field were irradiated daily with half the dose. This is entirely outside the scope of this paper, which is based solely on physical measurements.

One might wonder whether the relationship between port division and depth dose shown in this paper also exists in the super- and megavoltage range. Isodose curves obtained by others indicate that the transmarginal radiation of 1-million-volt beams is small and that of a 20-million-volt betatron beam is negligible. Until measurements similar to the ones here reported are made in this quality range of radiation, no definite conclusion on this point can be reached.

CONCLUSIONS

From the material presented the following conclusions applicable to x-rays of conventional deep-therapy quality can be drawn.

(1) In divided-port therapy, each small beam contributes a substantial amount of transmarginal radiation to regions irradiated by other small beams.

(2) This division improves the depth dose distribution in such a manner that an *increase* in the depth dose (over that of a single large beam) results in the marginal

[3] This rule probably cannot be applied to long narrow rectangles. The measurements which have shown its validity were confined to square ports and to broad oblong fields up to a length/width ratio of 2 (two squares in tandem). Not all of these measurements are published here.

TABLE VI: EXTENDED DEPTH-DOSE TABLE AS USED AT THE PRESBYTERIAN HOSPITAL

(Table G as published in the *Physical Foundations of Radiology* by Glasser, Quimby, Taylor and Weatherwax has been augmented by columns for 450, 600, 800 and 900 sq.cm. Bottom line illustrates choice of listed port combinations. Most of the r values listed in these additional columns have been obtained by extrapolation or interpolation; 16 were measured). Values apply to contiguous ports and ports separated from each other by less than 0.5 cm. For 1.0 cm. separation, see Discussion (p. 739).

DEPTH DOSE TABLE HVL: 1mm Cu TSD: 50 cm

Air Dose :		100	100	100	100	100	100	100	100	100	100	100	100	100	
Skin Dose :		124	127	130	136	140	143	144	146	149	150	152	156	158	
Area (Sq.cm):		28	38	50	100	150	200	225	300	400	450	600	800	900	
Depth	1 cm :	113	119	125	133	137	142	143	146	150	151	154	159	162	1 cm Depth
↓	2 cm :	98	105	112	122	127	132	134	137	143	144	149	153	155	2 cm ↓
	3 cm :	85	90	96	109	114	119	121	125	128	130	135	138	140	3 cm
	4 cm :	73	77	82	95	102	107	109	113	118	120	125	127	129	4 cm
	5 cm :	62	67	72	83	91	95	97	101	106	107	112	115	117	5 cm
	6 cm :	52	56	61	72	78	83	85	89	95	97	103	106	108	6 cm
	7 cm :	45	48	52	63	68	73	75	79	84	86	93	97	99	7 cm
	8 cm :	38	41	44	55	59	63	65	69	75	77	86	90	92	8 cm
	9 cm :	32	35	38	48	52	56	57	60	64	70	79	83	85	9 cm
	10 cm :	27	30	33	41	45	50	51	54	58	63	70	74	76	10 cm
	11 cm :	22	25	27	35	40	44	45	48	51	55	58	62	65	11 cm
	12 cm :	19	21	23	31	35	39	40	42	45	50	52	56	58	12 cm
	15 cm :	12	13	14	20	23	26	27	29	31	35	36	42	43	15 cm
	17 cm :	8	9	10	14	17	19	20	22	24	25	26	32	34	17 cm
	20 cm :	4	5	7	9	11	12	12	13	15	15	16	19	20	20 cm
Configuration and Division of Port :		6cm	7cm	8cm	10^2	10x15	$2x10^2$	15^2	2x10x15	$4x10^2$	$2x15^2$	2x15x20	$2x20^2$	$4x15^2$	

Instructions for use of Table VI

(1) Add the areas of all neighboring entrance ports and apply the sum to line 3 of this table.
(2) Select that column in Table VI which is labeled with this figure or lies nearest to it.
(3) Follow this column down to the desired depth.
(4) The figure thus obtained is the percentage depth dose.
(5) Apply this figure to the known air dose and obtain thus the actual depth dose within the individual beam participating in the port division.

portions of the irradiated volume of tissue.

(3) An additional increase will, of course, be found in the narrow zone of overlap which occurs in the depth if the ports are contiguous. It must be noted that overlap radiation represents superimposition of components within the geometrical beams, rather than contribution from transmarginal radiation.

(4) If overlap is avoided by appropriate spacing of portals, the transmarginal radiation effect alone is still great enough to prevent a drop of the depth dose as compared to the undivided port.

(5) The effect of transmarginal radiation is so great that it is virtually impossible to "exempt" completely a deep-seated organ from irradiation if it is located in the interspace between two or more rather widely separated beams.

(6) Neglecting the effect of transmarginal radiation when dealing with more than one port through which radiation enters the body in approximately the same direction may result in a large error. This effect is pronounced enough to warrant its recognition in routine depth-dose calculations. In the range of depth, qual-

TABLE VII: DEPTH-DOSE TABLE

(Identical with Table VI except that the doses are expressed in r per 100 r on skin, with back-scatter. Figures obtained from Table VI by calculation and re-interpolation)

DEPTH DOSE TABLE (r per 100 r on skin) HVL: 1mm Cu TSD: 50cm.

AIR DOSE:	81	79	77	74	72	70	70	68	67	67	66	64	63	
SKIN DOSE:	100	100	100	100	100	100	100	100	100	100	100	100	100	(with back scatter)
AREA (Sq. cm):	28	38	50	100	150	200	225	300	400	450	600	800	900	
Depth 1cm:	91	94	96	98	98	99	99	100	101	101	101	102	103	1cm. Depth
2cm:	79	83	86	90	91	92	93	94	96	96	97	98	98	2cm
3cm:	68	71	74	80	82	83	84	85	86	87	88	89	89	3cm
4cm:	59	61	63	70	73	75	76	77	79	80	81	82	82	4cm
5cm:	50	53	55	61	64	66	67	69	71	72	73	74	74	5cm
6cm:	42	45	47	53	56	58	59	61	64	65	67	69	69	6cm
7cm:	36	38	40	46	49	51	52	54	56	57	60	62	63	7cm
8cm:	31	32	34	40	42	44	45	47	50	51	54	57	58	8cm
9cm:	26	28	29	35	37	39	40	41	44	46	50	53	54	9cm
10cm:	22	24	25	30	33	35	36	37	39	41	44	47	48	10cm
11cm:	18	20	21	26	29	31	32	33	34	35	38	40	41	11cm
12cm:	15	17	18	23	25	27	28	29	30	31	34	36	37	12cm
15cm:	9	11	13	15	17	18	29	20	21	22	24	26	27	15cm
17cm:	6	7	8	10	12	13	14	15	16	17	19	21	22	17cm
20cm:	3	4	5	7	8	8	8	9	10	10	11	12	13	20cm
Configuration and Division of Port	6cm	7cm	8cm	10^2	10x15	$2x10^2$ or 10x20	15^2	2x10x15 or 15x20	$4x10^2$ or 20^2	$2x15^2$	2x15x20	$2x20^2$	$4x15^2$	

Instructions for use of Table VII

Proceed with Steps 1 to 4 as for Table VI and (5), having obtained the percentage depth dose figure on Table VII, apply it to the skin dose for the *total area* (not the individual port) and obtain thus the actual depth dose within the individual beam participating in the port division.

(6) Obtain the skin dose for the total area from line 2 of Table VI (not VII) or from your machine-setting chart. If neither is practical, derive it from the known skin dose of the individual port by this rule of approximation: doubling the area increases the skin dose by 5 per cent, quadrupling by 10 per cent, enlarging it sixfold by 12 per cent and eightfold by 15 per cent.

ity, and distance tested, actual values may be 15 to 76 per cent higher than the calculations if this radiation is disregarded, depending on depth and number of beams.

(7) No measurements have been made at the surface of the phantom, but it is reasonable to assume that the skin dose is influenced by transmarginal radiation in the same manner as the depth dose, though to a lesser degree. Conventional depth-dose tables for the above range of quality and distance indeed indicate that doubling the area of the entrance port increases the skin dose by 3 to 5 per cent, whereas quadrupling raises it by 8 to 10 per cent regardless of absolute size.

(8) The user of conventional depth-dose tables can correct for the error described in paragraph (6), in adequate approximation, by selecting from the tables depth-dose values applying to an entrance port which is as large as the sum of all adjacent portals through which radiation passes in approximately the same direction.

(9) Correction for transmarginal radiation can be made in this manner only from tables in which the depth dose is expressed in percentage of the air dose. In those tables in which the depth dose is expressed in percentage of skin dose, calculations have to be based on a theoretical skin dose which is correct for the total area of all neighboring entrance ports and which is larger than the skin dose of the participating individual small beams. Extrapolation is impractical from tables of this type.

(10) The relative effect of transmarginal radiation on the total depth dose is essentially independent of the total size of the ports. It depends largely on the

ratio between the area of the small individual entrance port and the area representing the sum of all participating ports.

(11) Correction for transmarginal radiation can be made also by the use of a special extended depth-dose table which includes in its listing the port combinations likely to occur in clinical radiotherapy. It makes extrapolation for large areas unnecessary. Tables of this type can be made to express the depth dose in percentage of skin dose as well as in percentage of air dose. An example of each is shown in Tables VI and VII. These tables can also be used in the conventional manner for depth-dose calculation in undivided-port therapy and can supplant conventional tables fully if target-skin distance and half-value layer are as indicated.

APPENDIX

The following hypothetical clinical examples are given in order to illustrate how the material here presented might be applied to actual treatment cases. For simplicity's sake, a half-value layer of 1 mm. Cu and target-skin distance of 50 cm. are assumed for both cases.

Example 1: A four-year-old boy with generalized abdominal neuroblastoma was treated through four adjacent 10 × 10-cm. portals over the anterior abdomen and a like number over the posterior abdomen. Each of these eight portals had received six treatments of 200 r in air when the course had to be interrupted because of intercurrent complications. Sometime later the question arose whether treatments should be resumed and what dose the mid-plane of the abdomen had received thus far. The anteroposterior diameter of the abdomen was 12 cm.

According to conventional depth-dose tables, the percentage depth dose for a 10 × 10-cm. entrance port is 72 per cent of the air dose at a depth of 6 cm. A total air dose of 1,200 r (200 r × 6) was given to each of the 10 × 10-cm. fields. Neglecting transmarginal contribution, one finds that the mid-plane of the abdomen received 2,400 r × 72/100 = 1,728 r through two opposing ports.

If, however, one follows the rule discussed in this paper for including transmarginal radiation by selecting the depth dose listed in the table for a 400-sq. cm. (20 × 20 cm.) port (instead of the one for a 100-sq. cm. field), one arrives at a 6-cm. depth dose of 95 r per 100 r in air. This indicates that the depth dose to the mid-plane of the abdomen was actually 2,400 r × 95/100 = 2,280 r, *i.e.*, 32 per cent higher than the former calculation implied.

Conversely, if a depth dose of approximately 2,000 r to the abdominal mid-plane of this patient is contemplated, the treatment plan is influenced significantly by the inclusion of transmarginal radiation in the calculation. Neglecting it, one arrives at a required air dose of 1,389 r to each of the eight ports, *i.e.*, a total of 56 treatments, whereas its inclusion lowers the required air dose to 1,055 r or approximately 42 treatments if 200 r in air are given each time.

Example 2: A woman received a routine course of x-ray treatments to the pelvis after having undergone conventional radium treatment for a carcinoma of the cervix. Two 10 × 15-cm. ports separated from each other by 5 cm. were used anteriorly and posteriorly. The patient received 2,800 r/air (14 × 200 r/air to each of four portals, in 34 days). This gave a tumor dose of 2,632 r to the parametria in the mid-plane of the pelvis (anteroposterior diameter of pelvis, 20 cm.) at points 7.5 cm. lateral to the mid-line. In order to protect mid-line structures (bladder, rectum) from radiation the portals were separated by a distance of 5 cm., as stated above. This patient, however, received considerable transmarginal radiation to the mid-line structures, which can be calculated by consulting Table V. This table shows that the mid-point of the interspace at a depth of 10 cm. received 37 per cent of the air dose delivered to each port. Since two portal pairs were applied (one anteriorly and one posteriorly), the 10-cm. depth dose at the mid-point of the interspace was 2 × 37 × 2,800 r/100 = 2,072 r. This dose was thus received by the pelvic mid-line structures in addition to gamma radiation delivered previously.

SUMMARY

Measurements have been presented which demonstrate that neighboring approximately parallel x-ray beams entering the body contribute a considerable amount of transmarginal radiation to each other. This raises the dose in the central axis of each neighboring beam significantly above that found in a single unaccompanied x-ray beam of equal cross section.

A simple method is described for approximating correct tissue doses from conventional depth-dose tables when this situation exists. It should be employed whenever a large port of entrance is divided into two or more smaller contiguous or nearly contiguous ports admitting the entrance of essentially parallel beams.

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SUMARIO

El Uso de Tablas Convencionales de Dosis Profunda en la Terapéutica Dividida en Puertas (Irradiación Transmarginal y Distribución de Dosis Profunda)

Terapéutica dividida en puertas es la clase de tratamiento que cubre una gran zona de piel a través de varias portezuelas por las que los haces de rayos X penetran en el cuerpo paralela o casi paralelamente entre sí. Esas puertas pueden ser contiguas o quedar a poca distancia. Con esa técnica, cada haz aporta una cantidad substancial de irradiación transmarginal a las regiones irradiadas por los haces cercanos. El olvido del efecto de esa irradiación transmarginal al computar la dosis profunda puede conducir a un error grave. En los límites de profundidad, calidad y distancia determinados por los AA., las cifras reales fueron de 15 a 76 por ciento más altas que los cálculos en que se desatendió dicho efecto, dependiendo la magnitud de la diferencia de la profundidad y del número de haces.

El que use las tablas convencionales de dosis a profundidad puede corregir el error descrito, escogiendo de las tablas (con tal que den la dosis a profundidad en porcentaje de la dosis al aire) cifras de la dosis a profundidad que se apliquen a una puerta de entrada de tamaño igual al de la de la suma de todas las puertas adyacentes por las cuales pasa irradiación en la misma dirección aproximada. Hay que ejecutar esta corrección siempre que una puerta grande esté dividida en dos o más portezuelas contiguas o casi contiguas que admiten haces esencialmente paralelos.

DISCUSSION

Anna Hamann, M.D. (Evanston, Ill.): The paper just presented calls attention to a mistake in dosage determination which is made frequently and has escaped discovery by many radiologists in spite of their use of isodose charts. Many of the conventional isodose charts furnish, for transmarginal irradiation, curves down to the level of 10 per cent of the surface dose only. If these charts are used for split-portal technic, they inform nicely as to the contribution of transmarginal irradiation which compensates for the marginal loss of dosage in an adjacent field and makes the dosage distribution in this technic relatively uniform. However, they fail to give any information on the contribution of transmarginal irradiation to the center of the adjacent field, the center always being a rather considerable distance away from the 10 per cent transmarginal isodose curve, even with relatively small portals. Extrapolation with adequate accuracy is impossible when, as customary, only two curves for transmarginal radiation are charted. As we physicians do not like to figure very much, we are prone to dismiss small fractions as negligible. In this instance we overlook the fact

that a small percentage of the surface dose may represent a considerable addition to the depth dose, especially when multiple split portals are used. The measurements of the authors show that, for h.v.l. 1 mm. Cu, 50 cm. F.S.D., and 10 × 15-cm. portal size, the contribution from one adjacent portal to the center of the other portal at 10 cm. depth is of the order of 20 per cent, while in a four-portal technic, the increase is slightly over 50 per cent. The clinical implications from mistakes of this magnitude in the estimation of dosage are evident. If a radiologist wanted to raise a conventional depth dose of 3,000 r to the magic figure of 6,000 r, the consequences would be deleterious. The more we are guided by figures instead of experience, the more important it becomes that dosage figures be as accurate as possible. Routine charting of depth-dose curves for transmarginal radiation below 10 per cent would be of help.

The authors have developed a relatively simple method for determining dosage in split-portal technic with permissible accuracy from conventional depth-dose tables for single portals by using the total irradiated area instead of the single portal as a guide. The question arises as to the limitations of the method by differences in spacing of the portals and by differences in the quality of radiation. For wide spacing and for more penetrating rays, a decrease in the side-scattering and therefore a decrease of the contribution of transmarginal radiation to the central parts of the adjacent fields should be expected. The importance of modifying standard depth-dose tables by additional data for split-portal technic, as shown by the essayists in their table for h.v.l. 1 mm. Cu and 50 cm. F.S.D., cannot be overemphasized. Only what is readily available is systematically recorded. It might be preferable to list the contributions for each split portal separately instead of the total, for easier daily recording when not all portals are treated on the same day. But this is a question of minor importance.

The essayists are to be congratulated for their contribution, which draws attention to a point of neglect and of potential hazard in a widely used technic of x-ray therapy.

Dr. Schwarz (*closing*): In reference to the limitations of this method as far as size and separation of ports are concerned, I can give only a "limited" answer: Extrapolation is limited by the largest port listed in conventional depth-dose tables. Since it is usually 20 × 20 cm. and since quadrupling is the largest practical increase of area, the limit would be 40 × 40 cm., which is a larger field than one is likely to encounter. Our actual measurements have not gone beyond 30 × 30 cm. or 900 sq. cm. The reason for this was not the existence of a theoretical limit but merely the fact that our phantom happened to be no larger than 30 × 30 × 30 cm. At this extreme size we noted that the increase in depth dose was somewhat less than predicted from the behavior of smaller ports. I believe that the reason for this lay in the fact that there was no phantom excess beyond the borders of a field of that size so that back-scatter was reduced. What would happen if one used a limitless phantom—as large as a table or as the whole room—I cannot say. Someone should be enticed into doing this kind of experimentation even though such a phantom would not be representative of the patient's body.

There is no limit to the degree of separation between ports—as one of the diagrams had shown—but the larger the separation the smaller is the dose increase. If one does not want to use special tables for various degrees of port separation but prefers to adhere to the above discussed extrapolation method, one can do so up to a limit of 1 cm. separation. In this case one should subtract 5 per cent from the calculated depth dose in two-port combinations and 10 per cent in four-port combinations. This will produce a fair approximation.

Radiation Dosage to the Female Genital Tract During Fluoroscopic Procedures[1]

H. STEPHEN WEENS, M.D., J. LUTHER CLEMENTS, M.D.[2] and JOHN H. TOLAN, B.S.

THE WIDESPREAD use of x-rays for diagnostic examinations requires greater attention to the hazards incident to radiologic procedures. Exposure of radiologists and technical personnel to scattered radiation has been assessed in several experimental and clinical studies, and proper protective recommendations have been made (1–5). Concurrently, observations regarding radiation dosages to patients undergoing a variety of diagnostic examinations have been recorded (2, 6–10). Exposure of the female genital tract has received particular attention because of the possible production of genetic damage by irradiation of the ovaries (11). Radiation levels to which an intrauterine fetus may be exposed during diagnostic procedures have also been given due consideration, with respect to induction of various fetal malformations (12).

Since radiation effects are largely dependent upon dosage, a more precise determination of exposure during diagnostic procedures appears necessary. Accurate data concerning radiographic exposures are readily available, inasmuch as actual working conditions are satisfactorily reduplicated in phantom experiments. The amount of radiation received by patients during fluoroscopic examinations is more difficult to determine, because of a number of variable factors, such as fluctuating field sizes, differences in duration of fluoroscopic observation, and changes in position of the patient. Information of this type appears desirable, since it is generally admitted that the exposure of the patient during fluoroscopy is among the highest involved in diagnostic procedures. For this reason, it was considered worth while to determine the dosage delivered to the female midpelvis and genital organs during actual fluoroscopic study of the gastrointestinal tract. The information was obtained by direct measurements with the aid of small ionization chambers.

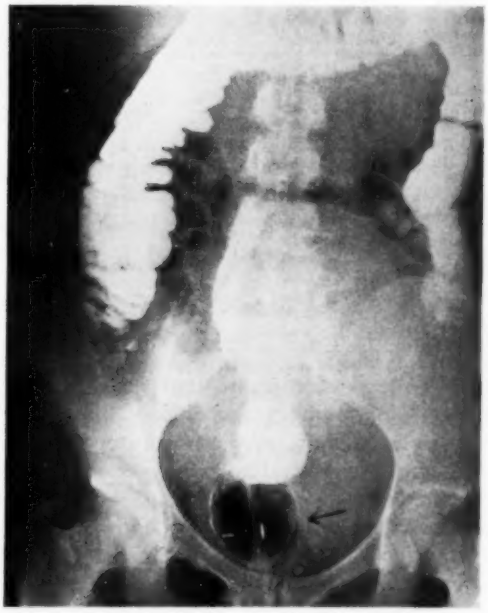

Fig. 1. Barium enema study. Position of midget ionization chamber indicated by arrow.

METHOD OF EXAMINATION

This study was performed on 15 female patients with various clinical conditions indicating examination of the large intestine by barium enema. In addition, dosage measurements were taken on 5 patients for whom routine gastrointestinal series were obtained. A small ionization chamber enclosed in a rubber finger cot was placed high in the lateral or posterior vaginal fornix of each patient (Fig. 1). This region is in close proximity to the ovaries and uterus, and the dosage to these areas should be approximately the same.

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TABLE I: DOSAGE TO THE FEMALE GENITAL TRACT ON FLUOROSCOPIC EXAMINATION OF THE COLON (BARIUM ENEMA)

Patient	A.P. Diameter Pelvis (cm.)	Fluoroscopic Time (min.-sec.)	No. of Spot Films of Sigmoid and Cecum	No. of 14 × 17-inch Abdominal Films	Dosage in r
1	17.5	2-36	4	3	0.4
2	13.0	2-45	4	3	0.8
3	22.0	2-10	4	3	0.4
4	20.0	3-57	4	3	0.6
5	12.0	3-30	3	3	0.4
6	17.0	2-5	4	4	0.4
7	15.0	2-9	1	3	0.3
8	17.5	1-50	3	4	0.5
9	20.0	1-21	1	4	0.5
10	28.0	3-10	4	4	0.4
11	18.0	1-57	3	0	0.2
12	16.5	1-23	2	0	0.6
13	21.0	2-20	1	0	0.1
14	20.0	2-24	4	0	0.3
15	17.0	3-44	6	0	0.4

The dosimeter used was a Landsverk type L-81 midget ionization chamber, chosen because it was immediately available and was suitable in physical dimensions and range for the measurements to be made. Since there was no complete information as to its physical characteristics, some effort was expended to ascertain if the readings obtained were a true indication of radiation exposure. The points of interest were energy dependence of the chamber, possible scale error, and possible effect of exposure rate. Other factors, such as peak kilovoltage, milliamperage, and time settings, were accepted at face value and assumed to be within the overall accuracy of the procedure.

The energy dependence of the chamber and possible scale error were considered together. The exposure rate of the x-ray apparatus was determined with a Victoreen Model 70 r-meter with 100 r bakelite wall chamber which had recently been recalibrated by the Radiological Equipment Section of the National Bureau of Standards. The Landsverk chamber was substituted for the Victoreen chamber and exposed for uniform increments of time as determined by an electric elapsed time-clock introduced into the x-ray control circuit. The readings obtained from the Landsverk chamber were then compared with the expected reading as determined by the Victoreen chamber, and a correction scale was prepared from the data. It was found that high percentage scale errors were present at low-exposure points, but that the Landsverk chamber gave a good indication of exposure at about three-quarters of full scale (1.5 r) for the particular quality of radiation used (85 kv.p., full-wave rectification).

The fluoroscopic examinations were carried out in the usual manner by three different examiners. The fluoroscopic time was measured by the interval timer mentioned earlier. The procedure was carried out at 85 kv.p. and 3.5 ma. (manufacturer's calibration), with 2.5 mm. of aluminum added filtration. With these factors, a dosage rate of 5.2 r/min. without backscatter is obtained on the top of the fluoroscopic table, at a target-to-table top distance of 46 cm.

Spot films of the sigmoid, splenic flexure, and cecal areas were taken. The average spot-film technic was 20 mas., at 85 to 95 kv.p., with field sizes measuring up to 8 × 10 inches. Usually two spot-film exposures of the sigmoid area and one to two of the cecal region were obtained.

In 10 of the patients receiving a barium enema, three to four large films of the abdomen were exposed on completion of the fluoroscopic examination, with exposure values of 20 to 60 mas. at 72 to 98 kv.p., according to the size and position of the patient (target-film distance 36 inches). All of the 14 × 17-inch films included the pelvic area and the dosimeter. In the remaining 5 patients studied by barium enema, radiation dosages were determined following the fluoroscopic and spot-film procedures in order to evaluate exposure originating from these examinations alone.

On completion of the radiologic studies, the dosimeter was removed; the readings were obtained and corrected in the prescribed manner, being recorded to the next highest 0.1 r.

DISCUSSION

Our results indicate that during routine barium enema studies the female mid-

pelvis received a radiation dosage between 0.3 and 0.8 r (Table I). As expected, the dosage during fluoroscopic examinations and film studies of the upper gastrointestinal tract appeared considerably lower (0.1 to 0.3 r) (Table II). These figures proved to be much smaller than we had anticipated. One should be aware, however, that the region of the uterus and ovaries is exposed only during a comparatively small fraction of the total fluoroscopic time. Thus, the position of the ionization chamber was usually not included in the limits of the spot roentgenograms depicting the sigmoid colon and cecum. Also, in the large survey films, though the area of the female genital tract was included, these structures were toward the periphery of the field, where dosage is appreciably smaller than along the axis of the central beam. The results in those patients in whom large abdominal films were omitted to evaluate the radiation exposure of fluoroscopy alone indicate that approximately one-third to one-half of the dosage exposure to the female tract on routine barium enema study is contributed by the taking of the large abdominal roentgenograms. This observation was confirmed in a study of radiation exposure associated with individual large films of the abdomen (Table III). Actually no correlation between radiation dosage and total fluoroscopic time could be established.

TABLE II: DOSAGE TO THE FEMALE GENITAL TRACT ON FLUOROSCOPIC EXAMINATION OF THE UPPER GASTROINTESTINAL TRACT (G.I. SERIES)

Patient	A.P. Diameter Pelvis (cm.)	Fluoroscopic Time (min.-sec.)	No. of Spot Films	No. of Films of Stomach (11 × 14 inches)	No. of Films of Abdomen (14 × 17 inches)	Dosage in r
1	18.0	5-34	4	4	1	0.1
2	20.0	5-19	8	4	1	0.2
3	18.0	4-3	6	4	1	0.3
4	17.0	5-36	4	3	1	0.1
5	22.0	7-46	12	4	1	0.2

It is realized that the experimental data described above are strictly applicable only to the apparatus and technical procedures employed in this study. On the basis of phantom measurements described below, however, it appears unlikely that radiation dosages in other departments would vary widely from those obtained by us.

TABLE III: DOSAGE TO THE FEMALE GENITAL TRACT ON INTRAVENOUS UROGRAPHY

(Focal-film distance 36 inches; 20–40 mas. per exposure at 74 to 88 kv.p.)

Patient	A.P. Diameter Pelvis (cm.)	No. of 14 × 17-inch Films	Dosage in r
1	15.0	4	0.2
2	17.0	4	0.1
3	20.0	4	0.2
4	15.0	4	0.2
5	15.0	4	0.2

It is well known that the roentgen outputs of fluoroscopes have shown a considerable range of values in various x-ray departments and physicians' offices (2, 10). To a large extent, this may be ascribed to variations in target-to-table top distances as well as to differences in the thickness of added filtration. In order to assess the importance of these factors, as well as their influence on surface and certain depth doses, a phantom experiment was undertaken. Because of the relative inaccessibility of the fluoroscopic tube, a radiographic tube was utilized. The radiation dosages were determined for two target-phantom surface distances. A distance of 46 cm. was selected to represent one of the newer types of fluoroscopic units, such as was used in the study described above. A distance of 30 cm. was chosen to simulate older types of fluoroscopic tables as well as upright fluoroscopic units commonly in use.

The phantom studies (Table IV) demonstrate that, in spite of fixed kilovolt and milliampere values (85 kv.p., 3.5 ma.), a large variation of roentgen output at the table top may exist if there are considerable differences in table top-target distances and filtration. Thus, in comparing a fluoroscopic technic with a target-table top distance of 30 cm. without added filtration and one with 46 cm. and 3 mm. added filtration, it will become evident that a dos-

TABLE IV: ROENTGENS PER MINUTE OF FLUOROSCOPY (85 kv.p., 3.5 ma.)

Added Filtration in mm. of Aluminum	46 cm. Target-to-Table Top Distance			30 cm. Target-to-Table Top Distance		
	Depth in Phantom			Depth in Phantom		
	Surface	10 cm.	20 cm.	Surface	10 cm.	20 cm.
0	20	1.9	0.20	60	3.2	0.28
1.0	13	1.6	0.17	34	2.7	0.24
2.0	8.4	1.4	0.15	24	2.3	0.21
3.0	6.3	1.2	0.13	17	1.9	0.18

age ratio of almost 10:1 will exist at the surface. At 10 cm. depth, however, which corresponds to the mid-pelvis, such differences become considerably smaller, while they are largely minimized at 20 cm., where the position of the fluoroscopic screen may be assumed.

From these observations, it may be concluded that in fluoroscopic examination high surface values do not necessarily reflect correspondingly high dosage rates in the mid-pelvis. Nevertheless, it is well demonstrated that surface dosages may be considerably reduced by proper filtration. This will also contribute to reduction of dosages within the body without significantly affecting dosages at the position of the fluoroscopic screen, as has previously been pointed out by various investigators (2, 3).

The radiation dosage to the female genital tract observed in our study is substantially lower than indicated by Ritter, Warren and Pendergrass (10). It should be stated, however, that these workers purposely gave maximum figures, applying to much longer fluoroscopic times. Also, the constantly shifting fluoroscopic field may not have been fully considered in their calculations. Our studies indicate, therefore, that the values proposed by Ritter and his co-authors involve a very large safety factor, as was intended.

In addition to dosage determinations in fluoroscopic examination, we have also conducted a small number of direct dosimetric measurements in patients undergoing radiographic examination by individual abdominal films (Table III) such as are obtained on intravenous urography. Our dosage range for single abdominal films to the mid-pelvis was found to fluctuate between 0.05 and 0.1 r per exposure. If differences in radiographic technic and the position of the ionization chamber toward the periphery of the field are considered, these values are in good agreement with phantom measurements by Braestrup (2) and Trout, Kelley, and Cathey (9).

A determination of these radiation dosages is of importance in relation to genetic damage resulting in an increased incidence of deleterious mutations. The approach to this problem is a difficult one, inasmuch as there is as yet no generally accepted understanding as to what may be considered a permissible increase of the normal spontaneous mutation rate. Though extensive observations have been made on genetic radiation effects in insects such as the fruit fly (13), comparatively little information is available with regard to higher animals (14). So far it has not been possible to predict genetic radiation effects in man with reference to various dosage levels. The dosage necessary to double the mutation rate in man has been estimated as high as 300 r and as low as 3 r (15).

The effect of radiation on the intrauterine fetus has recently received increasing attention. Russell and Russell (12) have shown, in animal experiments, that the fetus is unusually radiosensitive during certain early phases of gestation. This period of gestation corresponds approximately to the second to sixth week of human pregnancy. Rather large radiation dosages given during these periods will result in a very high incidence of fetal malformations. These authors state, however, that dosages as low as 25 r were noted to have a definite effect and that even smaller amounts may be expected to produce fetal malformations. Believing 25 r radiation exposure to the embryo to be in the range of fluoroscopic examinations, they advocate special precautions in the performance of radiologic procedures on female patients.

The dosage determinations described above indicate that fetal radiation exposure during fluoroscopy is markedly lower than was estimated by Russell and Russell. Nevertheless, until more accurate information concerning radiation effects on human mutation rates and fetal development become available, it will be wise to keep radiation exposure to the pelvic structures at a necessary minimum. It is hoped that the determinations described in this study, obtained under actual working conditions, may stimulate interest in evaluation of these low radiation levels as to possible genetic and fetal damage

SUMMARY

1. Radiation exposure of the female genital tract during fluoroscopic procedures has been studied by direct ionization measurements in the vaginal fornix.

2. In 20 patients on whom fluoroscopic procedures on the upper and lower gastrointestinal tract were performed, radiation exposure was found to be less than 1 r per examination.

3. The effect of radiation exposure of the female genital tract on the production of genetic damage and fetal malformations is briefly discussed.

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SUMARIO

La Dosis de Irradiación Recibida por el Aparato Genital Femenino Durante los Procedimientos Roentgenoscópicos

En 20 mujeres, se determinó, por medio de una cámara reducida de yonización introducida en el fórnix vaginal, la dosis de irradiación recibida en el aparato genital femenino durante el examen fluoroscópico de las porciones superior e inferior del tubo gastrointestinal. En 15 enfermas, en quienes se hicieron determinaciones durante un estudio con enema de bario, con radiografías instantáneas de las regiones del ciego y de la S ilíaca, la dosis de irradiación en la porción media de la pelvis quedó entre 0.3 y 0.8 r. En los estudios de la porción superior del tubo gastrointestinal, la dosis mesopelviana fué de 0.1 a 0.3 r.

Discútense los efectos genéticos de la exposición de los órganos genitales de la mujer a la radiación y la posible producción de anomalías fetales, deduciéndose que, hasta contar con información más exacta acerca de esos puntos, será prudente no exceder del mínimo necesario la exposición de los tejidos pelvianos.

EDITORIAL

Reflections on Hiatus Hernia and Related Problems

About a quarter of a century ago Forssell (16) published his classical description of hiatus hernia of the stomach and made the diagnosis appear as a straightforward radiological picture. Of equal importance, from another aspect, were Chevalier Jackson's (19) endoscopic observations on peptic ulcer of the oesophagus. But the relationship between hiatus hernia and peptic ulcer was not appreciated until the radiologist and endoscopist began to co-ordinate their findings. The example set by Clerf and Manges (9) and the entry of the thoracic surgeon into the field have brought so much to light that this clinical entity, once regarded as rare, now equals in frequency peptic ulcer of the stomach. Great strides have been made, much has been learned, but much remains to be learned or revised.

When a disease is newly discovered, it is usually in an advanced state, and peptic ulcer of the oesophagus is no exception. It is not intended, however, to imply that this lesion is a recent discovery for, as Barrett (5) has reminded us, pathologists have recorded many cases which have come to autopsy following perforation into the mediastinum or great vessels. Almost all the original cases to be published by clinicians (3, 7, 14, 15, 17, 18, 23) were characterised by dysphagia, and in many there was a severe degree of stenosis. In consequence, the radiological picture of a stenosis immediately above a pouch of herniated stomach was regarded as classical (20).

The gastric hernia was considered to be the result of a congenital shortening of the oesophagus (7), a theory which led to one of the early controversies, for an analysis of cases showed the incidence of hernia to be greatest in the fifty-to-seventy-year age group. Furthermore, the chronic inflammatory changes in the oesophageal wall following peptic erosion appeared by cicatrisation to produce some shortening of the oesophagus (3). Now it is generally accepted that most hernias are acquired: that they are related to the adiposity of middle age and to a hypersthenic build, and that there is a greater incidence in women, probably associated with the physical changes of pregnancy (26). It is possible, however, that the story is still incomplete, for experimental work in animals has shown that, on stimulation of the vagus through abdominal viscera, the lower oesophagus contracts, dragging up the gastric fundus into the thorax (11). Some support for this hypothesis might be found in the return to normal anatomical relationships which has been observed in children after medical treatment (8). Furthermore, there may be something in the incidence of coexisting cholecystitis or duodenal ulcer, although this has been denied by Brick (6).

More recently there appears to be a revival of the congenital theory, if one accepts Barrett's definition of the anatomical stomach. He contends that the stomach begins where the mucosa changes from stratified to columnar epithelium. There has never been any doubt that the junction is irregular and often lies in the terminal centimeter of oesophagus, but it may be found at a much higher level. Thus, an oesophagus partly lined with gastric mucosa may be the site of "gastric" ulcers. Barrett suggested that such ulcers were liable to the same complications as those in the stomach and duodenum, and concluded that the earlier cases of death due to oesophageal perforation, recorded by pathologists, were the sequelae of

gastric ulcer in the lower oesophagus. This entity is quite different from peptic ulceration of squamous epithelium, so ably described by Allison (1) as reflux oesophagitis, in which stenosis becomes the chief complication. Recently (2) it has been made clear that both lesions may occur in the same patient, and the recognition of such a combination must be of immense importance in assessing surgical treatment.

The original radiological picture of a combination of oesophageal stenosis and a gastric hernia has long been left behind, and the search for the early case has become intensive. Thus the efforts of radiologists are directed more and more to test the function of the cardia during routine gastrointestinal examination, with the result that a surprising number of abnormalities are being recorded, some of which fit into a recognisable pattern, while others appear equivocal or contradictory; particularly is this true in paediatrics. With experience, one has been able to make certain observations and draw conclusions; some have stood the test of time, others have had to be radically amended. Most of the difficulties arise from our incomplete knowledge of the anatomy and physiology of the cardia. Moreover, we are not assisted by the many widely differing statements which appear in textbooks, and it is hard to know where the truth lies. Templeton (28), in an excellent summary of the anatomy and physiology of the cardia, brought to light many controversial opinions concerning the dilatations or ampullae which may be found at the lower end of the oesophagus. The radiological differentiation of an oesophageal ampulla and a gastric hernia was first based on anatomical criteria, for it was thought possible to distinguish oesophageal from gastric mucosa by the mucosal relief picture. Unfortunately this has proved to be unreliable when checked by the application of silver clips to the junctional mucosa, and it is of great importance that this fallacy should be remembered (21).

Having realised the limitation of anatomical diagnosis, the radiologists turned their attention to reflux from the stomach, and concluded that if it occurred freely there was a breakdown in the physiological mechanism of the cardia, and peptic erosion of the oesophagus would follow. Once again the limitations of radiology must be recognised, for early pathological changes in the mucosa defy detection. Even in advanced changes, the radiological picture may fail to illustrate the severity of the lesion as seen at endoscopy.

The emphasis now laid on reflux brings up again the problem of the cardia (27). Anatomists, physiologists, and surgeons have failed to give an explanation of its function and mechanism which is universally accepted (24). There have been theories of pinchcock action of the diaphragm coupled with slight kinking of the hiatal oesophagus. These are the most popular but, since reflux does not regularly occur with a right phrenic nerve crush and food can pass into the stomach unimpeded by ordinary respiration, an element of doubt arises. This may be dispelled by the recent theory that the right crus receives its nerve supply from both phrenic nerves (10). In other quarters much support has been given to a valve mechanism produced by the angle made by the oesophagus at its junction with the stomach. This angle is said to be maintained by the sling of oblique muscle fibers running down the lesser curve. Yet radiologically one may see in cases of pneumoperitoneum a complete disappearance of the normal gastric fundus, so that the oesophagus and stomach merge into one continuous tube like the primitive gut, but there is no reflux—not even with the complication of phrenic crush (25).

Suggestions have been put forward that a trick mechanism such as belching is responsible for reflux (12). While most impressive demonstrations of reflux will occur in an established case of sliding hiatus hernia, most patients who constantly belch during examination have no reflux. In fact, they appear to be straining to overcome a sphincter mechanism in the lower oesophagus to relieve the intragas-

tric pressure. It has also been recorded that reflux does not always occur freely even in the presence of sliding hernia, and conversely that it may occur even in the absence of a hernia. Some patients are known to produce a hernia by contracting the abdominal muscles, and yet they are able to exert such control that the radiologist cannot demonstrate it without their co-operation. In others reflux has been observed to occur with each act of swallowing (22).

Facts are few, and recently, by combining simultaneous pressure recordings with radiology and rapid photofluorography, Dornhorst, Harrison, and Pierce (13) have concluded that none of the accepted theories of the nature of the cardia and its function will bear experimental verification.

Most radiologists must be aware of the fickleness of hernia and reflux; for example, both may be demonstrated readily and yet on subsequent examination, even a few minutes later, the phenomena may be absent. It is conceivable, however, that the weight of the meal reduces the hernia and that, if there is a good hiatal musculature, the diaphragm is able to exert sufficient control to maintain the stomach within the abdomen. If this hypothesis is accepted, it is possible that fatigue after physical exertion might permit the hernia to recur. One such case has been observed, which provoked the suggestion that radiologists should conduct gastrointestinal examinations in the evening. But perhaps it is not wise to generalise from the particular.

Recognition of the consequences of reflux has solved hitherto unexplained postoperative complications. For example, peptic strictures have followed oesophagogastrostomies or plastic operations for cardiospasm, and erosion of the oesophagus from pancreatic juices has been found after total gastrectomy with a short jejunal loop. The sudden postoperative onset of oesophageal peptic ulcer in cases with severe vomiting is another pointer to the dangers of reflux. But perhaps too much emphasis has been placed on the causal agents, with neglect of the protective action of the body. The secretion of mucus is an important factor and has been found plentiful in cases of hiatus hernia with little or no oesophagitis; cases with severe erosion showed a deficiency of mucus. Furthermore, it has been established that the type of gastric mucosa which extends widely into the oesophagus has few peptic or oxyntic cells and there is little danger from the local secretion.

Structures other than the oesophagus are also intimately involved, and deficiencies in the crura may be the primary cause of herniation. Little is known about the phreno-oesophageal membrane; some deny its efficiency in anchoring the oesophagus and some are even sceptical about its existence. Wolf *et al.* (29) have recently used the term "fixation failure" to cover some of those difficult cases which appear to be neither fish, flesh, nor good red herring. The term is attractive but these authors appear to have reached the conclusion, which to us is inevitable, that the final diagnosis and assessment cannot be made without the careful co-ordination of endoscopy and radiology. If inflammatory changes are observed, then there are adequate grounds for instituting treatment. Without such concrete evidence, it is unwise to blame a small hiatus hernia for vague symptoms which earlier may have been attributed to peptic ulcer elsewhere, cholecystitis, or angina—to mention a few of the possibilities. It is perhaps not without significance that in the follow-up series symptomatic cure may be perfect, yet the hernia and reflux have both recurred.

Surely there is no field more inviting for research, not only by the radiologist but by the anatomist, physiologist, clinician, biochemist, and pathologist. In fact, the whole team should start afresh and establish what is true. In this way it may be possible to standardise terminology and put facts and proved theories on a permanent and acceptable foundation.

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FIFTH INTER-AMERICAN CONGRESS OF RADIOLOGY

The Fifth Inter-American Congress of Radiology will take place April 24–29, 1955, at the Shoreham Hotel in Washington, D. C. This is the first opportunity radiologists of this country have had to receive and entertain their Latin-American friends. It is anticipated that there will be a large attendance.

The Program Committee has well advanced plans for an exceptionally fine program, a prominent feature of which will be refresher courses both in English and in Spanish. Through the kindness of the United States Public Health Service there will be a day devoted to a refresher course in the radioisotopes, followed by a guided tour of the National Cancer Institute and of the National Cancer Hospital.

The fact that the Congress is immediately preceded by the Annual Meeting of the American Radium Society offers an opportunity for a fairly prolonged graduate course which, no doubt, many radiologists, both North American and Latin-American, will wish to embrace.

Numerous social events will be included in the program, with special attention to the entertainment of the ladies.

Mark your calendar now for April 1955 and help to make this the greatest Inter-American Congress held thus far. PHILIP J. HODES, M.D.

KANSAS RADIOLOGICAL SOCIETY

The newly elected officers of the Kansas Radiological Society are: President, Willis L. Beller, M.D., Topeka; Vice-President, G. Sherman Ripley, M.D., Salina; Secretary-Treasurer, A. M. Cherner, M.D., Hays, Kans.

TRI-STATE RADIOLOGICAL SOCIETY

A new organization, the Tri-State Radiological Society, has been formed to serve the interest and needs of radiologists in southern Indiana, the northwestern part of Kentucky, and southeastern Illinois.

The following officers were elected to serve in 1953–54: President, Dr. M. Fitzgerald, Evansville, Ind.; Vice-President, Dr. H. Elkins, Mt. Carmel, Ill.; Secretary-Treasurer, Dr. S. N. Tager, 219 Walnut St., Evansville 9, Ind.

Meetings are to be held the last Wednesday of October, January, March, and May, 8:00 P.M., at the Elks' Club in Evansville.

WESTCHESTER RADIOLOGICAL SOCIETY

At a recent meeting of the Westchester (New York) Radiological Society, Dr. Roy Duckworth of White Plains was elected President, Dr. Walter Brown of Mt. Kisco Vice-President, and Dr. Maynard G. Priestman, New Rochelle Hospital, New Rochelle, Secretary-Treasurer.

SOCIETY OF NUCLEAR MEDICINE

The first official annual meeting of the Society of Nuclear Medicine will be held May 29–30, at the Benjamin Franklin Hotel, Seattle, Wash. The organizational meeting of this new society was held in Spokane, Wash., Jan. 23, 1954, with twelve representatives from various areas in the Pacific Northwest in attendance. At that time an Executive Committee was elected, consisting of Thomas Carlile, President; Asa Seeds, President-elect; Rex Huff, 2418 Harvard N., Seattle, Wash., Secretary; Norman Holter, Treasurer.

The purpose of the organization is to promote discussion and communication of knowledge of nuclear phenomena as they apply or are likely to apply to the understanding and control of disease. Those interested in becoming charter members should make application to Milo Harris, M.D., 252 Paulsen Bldg., Spokane, Wash. Abstracts of papers for presentation at the first meeting should be sent to William H. Hannah, RR 2, Bremerton, Wash.

Books Received

Books received are acknowledged under this heading, and such notice may be regarded as recognition of the courtesy of the sender. Reviews will be published in the interest of our readers and as space permits.

ROENTGENOGRAPHIC TECHNIQUE. A MANUAL FOR PHYSICIANS, STUDENTS, AND TECHNICIANS. Fourth edition. By DARMON ARTELLE RHINEHART, A.M., M.D., F.A.C.R., Emeritus Professor of Anatomy and Roentgenology, University of Arkansas; Honorary Member American Society of X-Ray Technicians; Honorary Registered X-Ray Technician; Roentgenologist to St. Vincent's Infirmary and the Missouri Pacific Hospitals, Little Rock, Ark. A volume of 454 pages, with 216 illustrations. Published by Lea & Febiger, Philadelphia, 1954. Price $8.50.

AN ATLAS OF CONGENITAL ANOMALIES OF THE HEART AND GREAT VESSELS. By JESSE E. EDWARDS, B.S., M.D., THOMAS J. DRY, B.A., M.A., M.B., Ch.B., M.S., ROBERT L. PARKER, M.D., M.S., F.A.C.P., HOWARD B. BURCHELL, M.D., PHD., EARL H. WOOD, M.D., PH.D., and ARTHUR H. BULBULIAN, M.S., D.D.S., F.A.C.D., of the Mayo Clinic and Mayo Foundation. A volume of 203 pages, with numerous illustrations,

graphs, and sketches. Published by Charles C Thomas, Springfield, Ill., 1954. Price $13.50.

DIE HALSWIRBELSÄULE. PATHOLOGIE UND KLINIK. By DR. MED. GERHARD EXNER, Privatdozent für Orthopädie an der Universität Marburg/Lahn mit einem Geleitwort von Prof. Dr. H. C. GEORG HOHMANN, München. A monograph of 140 pages, with 42 illustrations. Published by Georg Thieme Verlag, Stuttgart, 1954. Distributed in the United States and Canada by the Intercontinental Medical Book Corporation, New York. Price $5.15.

SCHICHTBILDER VON BRONCHIALVERÄNDERUNGEN BEI DER LUNGENTUBERKULOSE. By DR. MED. H. BLAHA, Assistent der Spezial-Lungenklinik in Hemer/Westfalen. A monograph of 113 pages with 86 illustrations. Published by Georg Thieme Verlag, Stuttgart, 1954. Distributed in the United States and Canada by the Intercontinental Medical Book Corporation, New York. Price $4.30.

RÖNTGENANATOMISCHE GRUNDLAGEN DER LUNGENUNTERSUCHUNG. By F. KOVÁTS, JR., Primarius des Tuberkulose-Forschungsinstitutes, and Z. ZSEBÖK, Dozent an der II. Chir. Universitätsklinik. A volume of 288 pages, with 311 illustrations. Published by Akadémiai Kiado, Budapest, 1953.

TRAITÉ TECHNIQUE DE TOMOGRAPHIE OSSEUSE. By ROBERT HERDNER. Preface by Professeur D. PETIT-DUTAILLIS. A volume of 407 pages, with 338 illustrations. Published by Masson et Cie, Paris, 1953. Price 4,600 fr.

CONFRONTATIONS RADIO-ANATOMO-CLINIQUES. Fascicule V. Published under the direction of M. CHIRAY, R. A. GUTMANN, and J. SENEQUE. A volume of 68 pages, with 135 illustrations. Published by Masson et Cie, Paris, 1954. Price 1,800 fr.

LA PRATIQUE DU RADIODIAGNOSTIC CLINIQUE. POSITIONS ET TECHNIQUES. By P. BUFFARD, Charge de Cours à la Faculté de Médecine, Radiologiste des Hôpitaux de Lyon, et L. CROZET, Radiologiste de l'Hôpital de Vienne. Avec la collaboration de M. GOYON. Préface du Doyen HENRI HERMANN. A monograph of 246 pages, with 221 illustrations. Published by G. Doin & Cie, Éditeurs, Paris, 1953. Price 2,900 fr.

Book Reviews

L'OESOPHAGE EN CARDIOLOGIE. ETUDE RADIOLOGIQUE DE L'OESOPHAGE DANS LES CARDIOPATHIES CONGÉNITALES ET ACQUISES. By MARCEL SEGERS, Chargé de Cours à l'Université de Bruxelles, and MARCEL BROMBART, Chef du Service de Radiodiagnostic à la Clinique C. de Paepe. Preface by PR. CH. LAUBRY. A monograph of 200 pages, with 135 illustrations. Published by Masson et Cie, 120, Boulevard Saint-Germain, Paris (VI°), 1953. Price 1,248 fr.

A rather extensive account of the use of the esophagram in studying patients with diseases of the heart and aorta has been undertaken in this monograph. Attention is called to the fact that the left atrium may impinge upon the esophagus during expiration, and that this should not be considered to be diagnostic of left atrial enlargement. The authors agree with Kjellberg that early left atrial enlargement is best shown in the recumbent position.

Various diseases of the heart and congenital anomalies of the aorta are covered in the later chapters. A fairly extensive bibliography is given. The work constitutes an interesting review of the significance of the opacified esophagus in cardiology.

DIE BEWEGUNGSBESTRAHLUNG. By Dr. F. WACHSMANN, Dozent für medizinische Physik, and Dr. G. BARTH, Dozent für Strahlenheilkunde und physikalische Therapie, Universität Erlangen. A volume of 192 pages, with 124 illustrations. Published by Georg Thieme, Stuttgart, 1953. Agents for U. S. A., Intercontinental Medical Book Corporation, New York. Price DM 36.00.

Increasing interest and work in moving-field therapy is aptly demonstrated by the publication of a monograph devoted to its various modalities.

The first chapter of this treatise by Wachsmann and Barth deals with the history, technical and physical development, fundamental physical and clinical aspects, and various types of moving-field therapy in general.

The second chapter is concerned with rotational irradiation. It describes the different machines and takes up the planning and technic of the treatments, dosage problems, indications, and clinical aspects. The authors advocate continuous fluoroscopic control to insure correct positioning during irradiations and therefore prefer vertical rotation of the patient to rotation around his recumbent form. They also prefer 200-kv. to the higher energies. Rotational therapy is indicated when the lesion is small, or long and narrow, and when it is situated in or close to the body axis.

The third chapter is concerned with the pendular type of moving-field therapy. The various equipments and their application are discussed in detail, as in the previous chapter. The pendular radiation may be used on recumbent patients. It may be employed also when the tumor is more extensive and is eccentrically located.

In the fourth chapter convergent beam therapy is discussed. With this technic, the tube rotates in

spiral form over a spherical segment, and the beam is continuously directed to the rotational axis point of the sphere. This method was developed and worked out by the authors, who are enthusiastic about its practical application. It may be used in two forms: (1) One convergent field should be used for medium-sized, moderately deep-seated tumors and for small lesions situated in the deeper structures. (2) Multiple cross-firing convergent beams may be employed for irradiation of extensive, deep-seated tumors.

Rotational therapy using supervoltage radiation is discussed in the fifth chapter. The various supervoltage equipments are briefly described and the fundamentals of supervoltage radiation are also considered. Some of the statements made here are highly questionable, *e.g.*, the statement that the energy increase above 12–15 mev is "senseless and deleterious."

A rather complete bibliography (196 references) concludes the book, with adequate references to the American literature.

The monograph is directed mainly to the physical, mechanical, and technical aspects of moving-field therapy, and in lesser degree to the clinical-practical aspects. Within these limits the book is comprehensive. It considers adequately American conditions, based on the personal American experiences of the physicist author. The style is stimulating and lively, and lends itself to easy reading. The book can be recommended to anyone who is interested in this special therapeutic field.

Union Station, Los Angeles. All rail lines leading to the Fortieth Annual Meeting of the Radiological Society of North America, Dec. 5 to 10, 1954, converge on this beautiful terminus.

RADIOLOGICAL SOCIETIES: SECRETARIES AND MEETING DATES

Editor's Note: Secretaries of state and local radiological societies are requested to co-operate in keeping this section up-to-date by notifying the editor promptly of changes in officers and meeting dates.

RADIOLOGICAL SOCIETY OF NORTH AMERICA. *Secretary-Treasurer*, Donald S. Childs, M.D., 713 E. Genesee St., Syracuse 2, N. Y.

AMERICAN RADIUM SOCIETY. *Secretary*, Robert E. Fricke, M.D., Mayo Clinic, Rochester, Minn.

AMERICAN ROENTGEN RAY SOCIETY. *Secretary*, Barton R. Young, M.D., Germantown Hospital, Philadelphia 44, Penna.

AMERICAN COLLEGE OF RADIOLOGY. *Exec. Secretary*, William C. Stronach, 20 N. Wacker Dr., Chicago 6.

SECTION ON RADIOLOGY, A. M. A. *Secretary*, Paul C. Hodges, M.D., 950 East 59th St., Chicago 37.

Alabama

ALABAMA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, J. A. Meadows, Jr., M.D., Medical Arts Bldg., Birmingham 5.

Arizona

ARIZONA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, R. Lee Foster, M.D., 507 Professional Bldg., Phoenix. Annual meeting with State Medical Association; interim meeting in December.

Arkansas

ARKANSAS RADIOLOGICAL SOCIETY. *Secretary*, Fred Hames, M.D., Pine Bluff. Meets every three months and at meeting of State Medical Society.

California

CALIFORNIA MEDICAL ASSOCIATION, SECTION ON RADIOLOGY. *Secretary*, H. R. Morris, M.D., 1027 D St., San Bernardino.

EAST BAY ROENTGEN SOCIETY. *Secretary*, Dan Tucker, M.D., 434 30th St., Oakland 9. Meets monthly, first Thursday, at Peralta Hospital.

LOS ANGELES RADIOLOGICAL SOCIETY. *Secretary*, George Jacobson, M.D., 1200 North State St., Los Angeles 33. Meets monthly, second Wednesday, Los Angeles County Medical Association Bldg.

NORTHERN CALIFORNIA RADIOLOGICAL CLUB. *Secretary*, H. B. Stewart, Jr., M.D., 2920 Capitol Ave., Sacramento. Meets last Monday of each month, September to May.

PACIFIC ROENTGEN SOCIETY. *Secretary*, L. Henry Garland, M.D., 450 Sutter St., San Francisco 8. Meets annually at time of California State Medical Association convention.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA. *Secretary-Treasurer*, Donald R. Laing, M.D., 65 North Madison Ave., Pasadena 1.

SAN DIEGO RADIOLOGICAL SOCIETY. *Secretary*, C. W. Bruner, M.D., 2456 Fourth Ave., San Diego 1. Meets first Wednesday of each month.

SAN FRANCISCO RADIOLOGICAL SOCIETY. *Secretary*, I. J. Miller, M.D., 2680 Ocean Ave., San Francisco 27. Meets quarterly, at the University Club.

SOUTH BAY RADIOLOGICAL SOCIETY. *Secretary*, James J. McCort, M.D., Santa Clara County Hospital, San Jose-Los Gatos Road, San Jose. Meets monthly, second Wednesday.

X-RAY STUDY CLUB OF SAN FRANCISCO. *Secretary*, Wm. W. Saunders, M.D., VA Hospital, San Francisco 21. Meets third Thursday at 7:45, Lane Hall, Stanford University Hospital.

Colorado

COLORADO RADIOLOGICAL SOCIETY. *Secretary*, Parker Allen, M.D., Children's Hospital, Denver. Meets monthly, third Friday, at University of Colorado Medical Center or Denver Athletic Club.

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary-Treasurer*, William A. Goodrich, M.D., 85 Jefferson St., Hartford 14. Meets bimonthly, second Wednesday.

CONNECTICUT VALLEY RADIOLOGICAL SOCIETY. *Secretary*, B. Bruce Alicandri, M.D., 20 Maple St., Springfield, Mass. Meets second Friday of October and April.

District of Columbia

RADIOLOGICAL SECTION, DISTRICT OF COLUMBIA MEDICAL SOCIETY. *Secretary*, Alvin C. Wyman, M.D., 5445 28th St., N.W., Washington. Meets third Wednesday, January, March, May, and October, at 8:00 P.M., in Medical Society Library.

Florida

FLORIDA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Hugh G. Reaves, M.D., Medical Arts Bldg., Sarasota. Meets in April and in October.

GREATER MIAMI RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Richard D. Shapiro, M.D., 541 Lincoln Road, Miami Beach. Meets monthly, third Wednesday, 8:00 P.M.

Georgia

ATLANTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Albert A. Rayle, Jr., M.D., 490 Peachtree St. Meets second Friday, September to May.

GEORGIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Robert M. Tankesley, M.D., 218 Doctors Bldg., Atlanta. Meets in November and at the annual meeting of the State Medical Association.

RICHMOND COUNTY RADIOLOGICAL SOCIETY. *Secretary*, Wm. F. Hamilton, Jr., M.D., University Hospital, Augusta. Meets first Thursday of each month.

Hawaii

RADIOLOGICAL SOCIETY OF HAWAII. *Secretary*, Philip S. Arthur, M.D., Suite 42, Young Hotel Bldg., Honolulu. Meets third Friday of each month.

Illinois

CHICAGO ROENTGEN SOCIETY. *Secretary*, Elbert K. Lewis, M.D., 6337 S. Harvard Ave., Chicago 21. Meets at the University Club, second Thursday of October, November, January, February, March, and April at 8:00 P.M.

ILLINOIS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Stephen L. Casper, M.D., Physicians and Surgeons Clinic, Quincy.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON RADIOLOGY. *Secretary*, George E. Irwin, Jr., M.D., 427 N. Main St., Bloomington.

Indiana

INDIANA ROENTGEN SOCIETY. *Secretary-Treasurer*, John A. Robb, M.D., 23 East Ohio St., Indianapolis. Annual meeting in May.

TRI-STATE RADIOLOGICAL SOCIETY (Southern Indiana, Northwestern Kentucky, Southeastern Illinois). Secretary-Treasurer, Stephen N. Tager, M.D., 219 Walnut St., Evansville 9, Ind. Meets last Wednesday, October, January, March, and May, 8:00 P.M., at the Elks' Club, Evansville, Ind.

Iowa

IOWA RADIOLOGICAL SOCIETY. *Secretary*, James T. McMillan, M.D., 1104 Bankers Trust Bldg., Des Moines. Meets during annual session of State Medical Society, and in the Fall.

Kansas

KANSAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, A. M. Cherner, M.D., Hays, Kansas. Meets in the Spring with the State Medical Society and in the Winter on call.

Kentucky

KENTUCKY RADIOLOGICAL SOCIETY. *Secretary*, David Shapiro, M.D., Veterans Administration Hospital, Louisville 6. Meets monthly, second Friday, at Seelbach Hotel, Louisville.

Louisiana

ORLEANS PARISH RADIOLOGICAL SOCIETY. *Secretary*, Joseph V. Schlosser, M.D., Charity Hospital of Louisiana, New Orleans 13. Meets second Tuesday of each month.

RADIOLOGICAL SOCIETY OF LOUISIANA. *Secretary-Treasurer*, J. T. Brierre, M.D., 700 Audubon Bldg., New Orleans.

SHREVEPORT RADIOLOGICAL CLUB. *Secretary*, W. R. Harwell, M.D., 608 Travis St. Meets monthly September to May, third Wednesday.

Maine

MAINE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Jack Spencer, M.D., Maine General Hospital, Portland 4. Meets three times a year—Spring, Summer, and Fall.

Maryland

BALTIMORE CITY MEDICAL SOCIETY, RADIOLOGICAL SECTION. *Secretary-Treasurer*, H. Leonard Warres, M.D., 2337 Eutaw Place, Baltimore 17. Meets third Tuesday, September to May.

MARYLAND RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, H. Leonard Warres, M.D., 2337 Eutaw Place, Baltimore 17.

Michigan

DETROIT X-RAY AND RADIUM SOCIETY. *Secretary*, E. F. Lang, M.D., Harper Hospital, Detroit 1. Meets first Thursday, October to May, at Wayne County Medical Society club rooms.

Minnesota

MINNESOTA RADIOLOGICAL SOCIETY. *Secretary*, John R. Hodgson, M.D., The Mayo Clinic, Rochester. Meets in Spring and Fall.

Mississippi

MISSISSIPPI RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John W. Evans, M.D., 117 N. President St., Jackson, Miss. Meets monthly, on third Tuesday, at 6:30 P.M., at the Rotisserie Restaurant, Jackson.

Missouri

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY. *Secretary*, James E. McConchie, M.D., First National Bank Bldg., Independence, Mo. Meets last Friday of each month.

ST. LOUIS SOCIETY OF RADIOLOGISTS. *Secretary*, Wm. B. Seaman, M.D., 510 South Kingshighway, St. Louis 10. Meets on fourth Wednesday, October to May.

Montana

MONTANA RADIOLOGICAL SOCIETY. *Secretary*, Grant P. Raitt, M.D., 413 Medical Arts Bldg., Billings. Meets annually.

Nebraska

NEBRASKA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, James F. Kelly, Jr., M.D., 816 Medical Arts Bldg., Omaha. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

New England

NEW ENGLAND ROENTGEN RAY SOCIETY. *Secretary*, Stanley M. Wyman, M.D., Massachusetts General Hospital, Boston 14. Meets monthly on third Friday, at the Harvard Club, Boston.

New Hampshire

NEW HAMPSHIRE ROENTGEN SOCIETY. *Secretary*, Albert C. Johnston, M.D., 127 Washington St., Keene.

New Jersey

RADIOLOGICAL SOCIETY OF NEW JERSEY. *Secretary*, Salomon Silvera, M.D., 921 Bergen Ave., Jersey City. Meets at Atlantic City at time of State Medical Society and midwinter in Elizabeth.

New York

BUFFALO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Mario C. Gian, M.D., 610 Niagara St., Buffalo 1. Meets second Monday, October to May.

CENTRAL NEW YORK ROENTGEN SOCIETY. *Secretary*, Dwight V. Needham, M.D., 608 E. Genesee St., Syracuse 2. Meets in January, May, and October.

KINGS COUNTY RADIOLOGICAL SOCIETY. *Secretary*, Solomon Maranov, M.D., 1450 51st St., Brooklyn 19. Meets fourth Thursday, October to April (except December), at 9:00 P.M., Kings County Medical Bldg.

NASSAU RADIOLOGICAL SOCIETY. *Secretary*, Frank Huber, M.D., 131 Fulton Ave., Hempstead, N. Y. Meets second Tuesday, February, April, June, October, and December.

NEW YORK ROENTGEN SOCIETY. *Secretary*, Jacob R. Freid, M.D., 1049 Park Ave., New York.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Donald H. Baxter, M.D., Albany Hospital, Albany. Meets in the capital area second Wednesday, October, November, March, and April. Annual meeting in May or June.

RADIOLOGICAL SOCIETY OF NEW YORK STATE. *Secretary-Treasurer*, Mario C. Gian, M.D., 610 Niagara St., Buffalo. Meets annually with the State Medical Society.

ROCHESTER ROENTGEN-RAY SOCIETY. *Secretary-Treasurer*, Henry H. Forsyth, Jr., M.D., 40 Meigs St., Rochester 7. Meets at Strong Memorial Hospital, 8:15 P.M., last Monday of each month, September through May.

WESTCHESTER RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Maynard G. Priestman, M.D., New Rochelle Hospital, New Rochelle, N. Y. Meets third Tuesday of January and October and at other times as announced.

North Carolina

RADIOLOGICAL SOCIETY OF NORTH CAROLINA. *Secretary*, Waldemar C. A. Sternbergh, M.D., 1400 Scott Ave., Charlotte 2. Meets in April and October.

North Dakota

NORTH DAKOTA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, H. Milton Berg, M.D., Quain & Ramstad Clinic, Bismarck. Meets in the Spring with State Medical Association; in Fall or Winter on call.

Ohio

OHIO STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, M. M. Thompson, Jr., M.D., 316 Michigan St., Toledo.

CENTRAL OHIO RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Howard W. Bangs, 1381 West Sixth Ave., Columbus 12. Meets second Thursday, October, December, February, April, and June, 6:30 P.M., Columbus Athletic Club, Columbus.

CLEVELAND RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Mortimer Lubert, M.D., Heights Medical Center Bldg., Cleveland Heights 6. Meets at 6:45 P.M. on fourth Monday, October to April, inclusive.

GREATER CINCINNATI RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Chapin Hawley, M.D., 927 Carew Tower, Cincinnati 2. Meets first Monday of each month, September to June, at Cincinnati General Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY. *Secretary*, W. S. Koller, M.D., 60 Wyoming St., Dayton. Meets monthly, second Friday.

Oklahoma

OKLAHOMA STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John R. Danstrom, M.D., Medical Arts Bldg., Oklahoma City.

Oregon

OREGON RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John Wayne Loomis, M.D., 919 Taylor Street Bldg., Portland 5. Meets monthly, second Wednesday, October to June, at 8:00 P.M., University Club, Portland.

Pacific Northwest

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Sydney J. Hawley, M.D., 1320 Madison St., Seattle 4. Meets annually in May.

Pennsylvania

PENNSYLVANIA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, James M. Converse, M.D., 416 Pine St., Williamsport 8. Meets annually.

PHILADELPHIA ROENTGEN RAY SOCIETY. *Secretary*, Herbert M. Stauffer, M.D., Temple University Hospital, Philadelphia 40. Meets first Thursday of each month at 5:00 P.M., from October to May, in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY. *Secretary-Treasurer*, Donald H. Rice, M.D., 4800 Friendship Ave., Pittsburgh 24. Meets monthly, second Wednesday, at 6:30 P.M., October to May, at Webster Hall.

Rocky Mountain States

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, John H. Freed, M.D., 4200 East Ninth Ave., Denver 7, Colo.

South Carolina

SOUTH CAROLINA RADIOLOGICAL SOCIETY. *Secretary-Treasurer*. William A. Klauber, M.D., Self Memorial Hospital, Greenwood. Meets with State Medical Association in May.

South Dakota

RADIOLOGICAL SOCIETY OF SOUTH DAKOTA. *Secretary-Treasurer*, Donald J. Peik, M.D., 303 S. Minnesota Ave., Sioux Falls. Meets during annual meeting of State Medical Society.

Tennessee

MEMPHIS ROENTGEN CLUB. *Secretary*, Harvey Thompson, M.D., 899 Madison Ave. Meets first Monday of each month at John Gaston Hospital.

TENNESSEE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, George K. Henshall, M.D., 311 Medical Arts Bldg., Chattanooga 3. Meets annually with State Medical Society in April.

Texas

DALLAS-FORT WORTH RADIOLOGICAL CLUB. *Secretary*, Otto H. Grunow, M.D., 650 Fifth Ave., Fort Worth 4, Texas. Meets monthly, third Monday 6:30 P.M., at the Greater Fort Worth International Airport.

HOUSTON RADIOLOGICAL SOCIETY. *Secretary*, Harry Fishbein, M.D., 324 Medical Arts Bldg., Houston 2.

SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY. *Secretary*, Hugo F. Elmendorf, Jr., M.D., 730 Medical Arts Building, San Antonio 5, Texas. Meets at Brook Army Medical Center, the first Monday of each month.

TEXAS RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, R. P. O'Bannon, M.D., 650 Fifth Ave., Fort Worth. Next meeting Jan. 29–30, 1954, Dallas.

Utah

UTAH STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Angus K. Wilson, M.D., 343 S. Main St., Salt Lake City 1. Meets third Wednesday, January, March, May, September, November.

Virginia

VIRGINIA RADIOLOGICAL SOCIETY. *Secretary*, P. B. Parsons, M.D., 1308 Manteo St., Norfolk 7.

Washington

WASHINGTON STATE RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, Eva L. Gilbertson, M.D., 1317 Marion St., Seattle 4. Meets fourth Monday, September through May, at 610 Pine St., Seattle.

West Virginia

WEST VIRGINIA RADIOLOGICAL SOCIETY. *Secretary*, W. Paul Elkin, 515–519, Medical Arts Bldg., Charleston. Meets concurrently with annual meeting of State Medical Society, and at other times as arranged by Program Committee.

Wisconsin

MILWAUKEE ROENTGEN RAY SOCIETY. *Secretary-Treasurer*, Jerome L. Marks, M.D., 161 W. Wisconsin Ave., Milwaukee 1. Meets monthly on fourth Monday at the University Club.

SECTION ON RADIOLOGY, STATE MEDICAL SOCIETY OF WISCONSIN. *Secretary*, Abraham Melamed, M.D., 425 E. Wisconsin Ave., Milwaukee 2. Meets in October with State Medical Society.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE. Meets first and third Thursday at 4 P.M., September to May, Service Memorial Institute.

WISCONSIN RADIOLOGICAL SOCIETY. *Secretary-Treasurer*, W. W. Moir, M.D., Sheboygan Memorial Hospital, Sheboygan.

Puerto Rico

ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA. *Secretary*, Rafael A. Blanes, M.D., Box 9724 Santurce, Puerto Rico.

CANADA

CANADIAN ASSOCIATION OF RADIOLOGISTS. *Honorary Secretary-Treasurer*, D. L. McRae, M.D., *Assoc. Hon. Secretary-Treasurer*, Guillaume Gill, M.D. *Central Office*, 1555 Summerhill Ave., Montreal 25, Quebec. Meets in January and June.

LA SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALES. *General Secretary*, Ls Ivan Vallée, M.D., Hôpital Saint-Luc, 1058 rue St-Denis, Montreal 18. Meets third Saturday of each month.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPÍA DE CUBA. *Secretary*, Dr. Rafael Gómez Zaldívar. Offices in Hospital Mercedes, Havana. Meets monthly.

MEXICO

SOCIEDAD MEXICANA DE RADIOLOGÍA, A. C. *Headquarters*, Calle del Oro, Num. 15, Mexico 7, D. F. *Secretary General*, Dr. Eugenio Toussaint. Meets first Monday of each month.

PANAMA

SOCIEDAD RADIOLÓGICA PANAMEÑA. *Secretary-Editor*, Luis Arrieta Sánchez, M.D., Apartado No. 86, Panama R de P.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Head and Neck

The Chest

The Digestive System

The Musculoskeletal System

Gynecology and Obstetrics

The Genitourinary System

The Blood Vessels

Technic and Apparatus

Miscellaneous

RADIOTHERAPY

RADIOISOTOPES

RADIATION EFFECTS

ROENTGEN DIAGNOSIS

THE HEAD AND NECK

The Relative Accuracy of Electroencephalography, Air Studies and Angiography in a Series of Two Hundred Mass Lesions. F. A. Martin, J. E. Webster, and E. S. Gurdjian. J. Neurosurg. **10**: 397–403, July 1953.

The relative value of electroencephalography, pneumoencephalography, ventriculography, and angiography as used in the diagnosis of 200 cases of mass intracranial lesions is discussed.

Electroencephalograms were recorded preoperatively in 198 patients. Only 93 (47 per cent) of the lesions could be localized to a discrete area. False foci were indicated in 45 cases. It is concluded that this type of examination is useful only as an aid to other diagnostic methods.

Pneumoencephalograms were obtained in 82 cases, and 30 of the tumors (36 per cent) were localized. An exchange of 100 to 150 c.c. of air for fluid was usually attempted. Accurate localization was made most often in cases of rapidly growing gliomas of the cerebral hemispheres and mid-line base lesions. These latter caused defects in the pattern of the basal cisterns.

Ventriculography was found to be the most useful method in the localization of an intracranial mass, being accurate in 90 per cent of 96 cases so studied. It is the only procedure that is helpful in the diagnosis of posterior fossa masses, with the exception of vertebral angiography. It is also the method of choice in cases with intracranial hypertension.

Carotid angiography was performed in 57 patients, and 31 masses were exactly localized. Lesions in the anterior two-thirds of the cerebral hemispheres and those occurring in the optic and supra-optic regions are best studied by this method.

The authors conclude that no single method is adequate in the localization of all types of intracranial mass lesions. The type of examination used will depend on the individual patient and the examiner.

Five tables. SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Ventricular Displacement and Electroencephalographic Focus in Multiple Sclerosis. P. A. Lindstrom. Arch. Neurol. & Psychiat. **70**: 254–259, August 1953.

A case is reported demonstrating that ventricular displacement, simulating that associated with brain tumor, may occur in multiple sclerosis. The patient, aged 29, had served in the Air Corps as a gunner. For five years prior to the present study he had poor hearing, spells of mild vertigo, and occasional unsteadiness, slurred speech, and clumsiness of one limb or another. He had experienced episodes of paresis of the right hand and the feet and recently had become violently dizzy, with sudden paralysis of the left extremities, diffuse headache, nausea, and vomiting. There was some past history of epileptiform attacks but no grand mal seizures.

Examination showed clear sensorium, some deafness, horizontal nystagmus bilaterally, temporal pallor of eyegrounds, paralysis and hypesthesia of the left extremities, increased deep reflexes, a Babinski sign on the left, and absence of abdominal reflexes. The spinal fluid was clear and colorless, under 210 mm. pressure, with two white cells per cubic millimeter, 68 mg. protein per 100 c.c., and a flat gold curve. The electroencephalogram showed a pronounced slow-wave focus in the right fronto parietal area.

A pneumoencephalogram revealed displacement of the lateral ventricles to the left and a marked distortion of the mid-portion of the right lateral ventricle, with some compression of the right temporal horn, as though there were a tumor centering in the right rolandic area. If a tumor was present, a large hemorrhage into it could have produced the recent sudden onset of symptoms. Under such circumstances, however, the patient should have been drowsy and the spinal fluid should have shown evidence of the hemorrhage. A gradually enlarging tumor would hardly account for the sudden paralysis, while a diffusely infiltrating tumor would probably not produce the focal signs and symptoms. It was therefore assumed that the ventricular shift was due to a localized swelling of the brain caused by an acute sclerotic plaque, and not a tumor. Craniotomy was not performed.

The patient gradually recovered and at the time of the report was fairly well except for slight weakness in the left arm and leg. He was gainfully employed. An electroencephalogram and pneumoencephalogram obtained ten weeks after the initial studies showed only slight symmetrical dilatation of the lateral ventricles, which were in normal position. The final diagnosis was multiple sclerosis.

Two roentgenograms; 1 line drawing.
BERNARD S. KALAYJIAN, M.D.
Detroit, Mich.

Ventriculographic Changes in Cysticercosis of the Brain. D. Govinda Reddy and B. Ramamurthy. Brit. J. Surg. **41**: 11–12, July 1953.

Cysticercus infestation may occur in any part of the brain, the leptomeninges and ventricles being involved most frequently. The diagnosis is chiefly dependent upon the clinical history. The associated presence of cysticerci in the subcutaneous tissue and somatic musculature strongly supports the suspicion of brain involvement. After calcification of the larvae, plain skull films may be helpful in locating the lesions.

The author describes a proved case of cysticercosis in a 12-year-old Hindu girl, with multiple hemispherical protrusions into the wall of the left lateral ventricle demonstrable on the ventriculogram. The ventricles were not dilated and were normal in position. It is suggested that a ventriculographic diagnosis of cysticercosis of the brain is possible if the cysts are located near enough to the ventricles to cause alteration in their outline.

Two roentgenograms; 1 photomicrograph.
C. M. GREENWALD, M.D.
Cleveland Clinic

Cerebral Angiography. Fundamentals in Anatomy and Physiology. Philip J. Hodes, Francisco Campoy, Helen E. Riggs, and Paul Bly. Am. J. Roentgenol. **70**: 61–81, July 1953.

Over 110 brains were injected with a suspension of barium in formalin, radiographed, and subsequently dissected for anatomical detail. Through this method of study the authors sought to determine (1) normal anatomical variations in the vascular pattern, (2) how

closely vessels of one half of the brain mirror those of the other half, and (3) the effect of improper positioning of the head on the roentgen appearance of normal vascular patterns. Methods of study included injection of the internal carotid artery, injection of the vertebral arteries following ligation of the posterior communicating vessels, radiography of the brain within the skull and after removal and sectioning of the hemispheres.

The most interesting anatomical findings concerned variations in the circle of Willis. Anomalies encountered on angiography were reduplication of the anterior cerebral artery and instances of two or three middle cerebral vessels rather than one arising from the internal carotid artery. The basilar artery was observed to be inconstant in its length, level of bifurcation, and distance from the plane of the clivus. It rarely occupied a mid-line position. Asymmetry of the cerebellar arteries and posterior cerebral artery was found to occur more frequently than symmetry.

In addition to the above briefly mentioned anatomical variations, the authors review cerebral vascular anatomy, describe pathologic changes encountered in routine autopsy material, and discuss cerebral vascular physiology. The latter material is drawn from the works of Kety and Schmidt (see, for example, Kety, S. S.: The Quantitative Measurement of Cerebral Blood Flow in Man, Methods in Medical Research. Chicago, Year Book Publishers, Vol. I, 1948. Schmidt, C. F.: The Cerebral Circulation in Health and Disease, Springfield, Ill., Charles C Thomas).

DORIS E. PIPKIN, M.D.
University of Louisville

Vertebral Angiography in Cerebellar Haemangioma. Olle Ollson. Acta radiol. **40:** 9–16, July 1953.

Seven cases of hemangioma of the cerebellum are reported. Six of these were diagnosed by angiograms obtained by catheterization of the vertebral artery. One failed to show radiologic changes. All patients were operated upon and the diagnosis was verified. A brief review of previous reports is presented.

These tumors are rarely multiple when occurring in the cerebellum. In 1 of the author's cases there was a second angioma in the medulla oblongata. Histologically the vascular structures vary from small capillaries to large cavernous spaces.

The angioma most frequently presents as a small network of entangled vessels in the vicinity of the posterior fossa. One of the author's cases, showed a large cystic area within the lesion, producing a ring-like appearance.

The hemangioma is more distinct in the late or venous phase of the angiogram, after overlying arterial densities have diminished. One of the cases reported was visualized only in the arterial phase. Vascular displacement was slight or absent in all cases.

Eleven roentgenograms.

NEIL E. CROW, M.D.
University of Arkansas

Roentgenologic Recognition of Habenular Calcification as Distinct from Calcification in the Pineal Body. Its Application in Cerebral Localization. Herbert M. Stauffer, Leo B. Snow, and Andrew B. Adams. Am. J. Roentgenol. **70:** 83–89, July 1953.

The authors identified habenular calcification in 89 out of 187 skull roentgenograms showing calcification in the pineal region. In 49 of these cases there was associated pineal calcification.

The habenular calcium is apparent in lateral skull roentgenograms as a "C"-shaped fleck in the pineal region. It represents calcification in the taenia habenulare and is separated from the pineal body by the habenular commissure. In encephalograms and ventriculograms it appears to lie in direct contact with the air in the posterior portion of the third ventricle, anterior to the habenular commissure, between the pineal recess and the suprapineal recess. In this series of observations the habenular calcification was, on an average, 5.8 mm. anterior to the center of the pineal body calcification.

Because of its highly specific localization, the characteristic habenular calcification provides a more accurate reference point in relation to neighboring brain structures than the much more variable pineal calcification.

Five illustrations, including 2 roentgenograms and a chart showing distribution of habenular and pineal body calcifications with respect to the anteroposterior diameter of the skull.

J. F. BERRY, M.D.
Louisville General Hospital

Agenesis of the Corpus Callosum with Concomitant Malformations, Including Atresia of the Foramens of Luschka and Magendie. Eugene F. Van Epps. Am. J. Roentgenol. **70:** 47–60, July 1953.

The author repeats Davidoff and Dyke's summary of the characteristic findings in agenesis of the corpus callosum (Am. J. Roentgenol. **32:** 1, 1934):

1. Abnormally great separation of the lateral ventricles.
2. Concave mesial margins and pointed dorsal margins of lateral ventricles.
3. Enlargement of the atria and temporal horns.
4. Elongation of interventricular foramens.
5. Dorsal extension and dilatation of the third ventricle.
6. Radial arrangement of mesial cerebral sulci.

A series of 12 cases of agenesis of the corpus callosum is presented: 4 discovered at autopsy and 8 diagnosed by means of air introduced either directly into the ventricles or injected into the spinal subarachnoid space. Enlargement of the cisterna magna was demonstrated in 5 cases. In 2 cases there was associated atresia of the foramens of Luschka and Magendie, though this was not recognized roentgenographically. In such cases the author believes, with Taggart and Walker (Arch. Neurol. & Psychiat. **48:** 583, 1942. Abst. in Radiology **41:** 29, 1943), that this atresia is the basic defect, producing an enlargement of the fourth ventricle with consequent underdevelopment of the cerebellum. An interesting associated roentgen finding is the failure of migration of the transverse confluent sinuses to their normal adult position in the occipital bone. In one of the author's cases the transverse sinuses are noted to be in the posterior parietal bone on the scout films. This may be a new roentgen sign suggesting underlying posterior fossa pathology. In one case a cerebral angiogram aided in the diagnosis of absence of the corpus callosum.

Anomalies found in association with agenesis of the corpus callosum included: cleft palate and harelip in 2 cases; porencephaly in 1 case; teratoma arising from the hypophysis in 1 case; abnormal fourth ventricle in

2 cases; atresia of the aqueduct in 1 case; cortical atrophy in 4 cases; cavum septum pellucidum in 1 case; absence of the posterior arch and spinous process of the atlas in 1 case; lenticular opacities, microophthalmia, and optic atrophy in 1 case; atrophy of one side of the body in 2 cases.

Twenty roentgenograms; 1 photograph.

LAWRENCE A. DAVIS, M.D.
University of Louisville

Occipitalization of the Atlas. D. L. McRae and A. S. Barnum. Am. J. Roentgenol. **70**: 23–45, July 1953.

The authors report 25 cases of occipitalization of the atlas, also referred to as assimilation of the atlas to the occipital bone, a congenital bony deformity occurring at the foramen magnum. In order to make this diagnosis, one must show some degree of bony union between the skull and the atlas. In all of the authors' cases there appeared to be bony continuity between the anterior lip of the foramen magnum and the anterior arch of the atlas. In most of the cases the cortex of the anterior arch was continuous with that of the basiocciput and in some cases the medulla was also continuous. Atlanto-occipital joint spaces were demonstrated only 3 times. In at least 11 cases, one or both transverse processes were attached to both the occipital and the lateral mass of the atlas. An overexposed anteroposterior film of the skull was found to be the best method of demonstrating fusion or nonfusion of the transverse process. Some trace of the posterior arch of the first cervical was present in all but two cases. Most commonly it was represented by a bony fringe on the posterior edge of the foramen magnum, which was directed downward and inward, producing some constriction of the spinal canal. Occasionally the fringe was present only on one side. The second and third cervical vertebrae were fused in 17 of the 25 cases of this series.

There is a question in the mind of the authors whether occipitalization of the atlas *per se* is ever productive of symptoms or signs without encroachment on the bulb or cord by associated soft-tissue deformities or other bony malformation. Posterior displacement of the odontoid process of the axis, which occurred in 15 of the 25 cases appears to be the most frequent cause of symptoms due to bony malformation. Another frequent finding was a thickened band of dura posteriorly constricting the cord. In 3 of the 12 cases which were surgically explored, cerebellar tonsil herniation was associated with assimilation of the atlas. Platybasia occurred in 4 cases.

The presence of associated congenital abnormalities was noted in approximately 20 per cent of the series, including hypoplasia of the jaw, incomplete cleft of the nasal cartilage, hypospadias, cleft palate deformity of the external ear, cervical ribs, and incomplete rotation of the kidney with normal renal vessels. Of the 18 patients whose symptoms could be explained by the anomaly at the foramen magnum and upper cervical region, 6 related their complaints to injury. Seven of the 18 symptomatic cases had been previously misdiagnosed, most frequently as multiple sclerosis. The most pronounced symptoms, due to cervical cord compression, were weakness and ataxia of the lower extremities. Numbness or pain in the upper extremities was of frequent occurrence, and a few patients complained of occipital headache, blurring of vision, and diplopia. Objective findings consisted mostly of hyperreflexia, Babinski and Hoffmann reflexes, weakness, and other long tract signs in both the upper and lower limbs. Nystagmus was a frequent finding and hypesthesia was seen in the extremities.

The authors stress the importance of recognizing this anomaly in the presence of the syndrome described, because of the possibility of surgical correction.

Two tables list all of the cases in the series, with the various physical, x-ray, and surgical findings. An excellent discussion of the embryology of the upper cervical vertebrae and the occipital region is included.

Forty-two roentgenograms

CLAUDE D. BAKER, M.D.
Louisville General Hospital

Localized Thinning and Enlargement of the Cranium with Special Reference to the Middle Fossa. Arthur E. Childe. Am. J. Roentgenol. **70**: 1–21, July 1953.

Thirteen cases are presented of localized thinning of the skull combined with localized enlargement of the cranial cavity. All of the cases except one showed bulging of the outer table. The most frequent cause of this cranial thinning and enlargement was chronic subdural hygroma, considered by some to be the result of subdural hematoma following trauma with a tear in the arachnoid allowing escape of fluid into the potential subdural space. Other causes were cerebral agenesis, glioma, neurofibromatosis, intracranial aneurysms, and chronic subdural hematoma.

A tear in the arachnoid may allow accumulation of fluid outside the cerebrospinal fluid circulation and produce compression of the intracranial contents exactly like a subdural hematoma. The seven cystic hygromas in this series contained clear fluid and with a single exception had thin arachnoid-like walls.

The most common roentgenographic finding was an enlarged middle fossa. The roentgen picture in the single case of cerebral agenesis in this series was quite similar to that produced by chronic subdural hematomas and hygromas. In such cases pneumoencephalography may be required for differentiation. Of the two gliomas, only one, originating in childhood, produced notable deformity. When the tumor develops later in life, after the skull has ceased to grow, such changes are slight. Cerebral angiography led to diagnosis of the case of intracranial aneurysm included.

Most of the patients in whom roentgenographic changes are present are children or young adults with an old history of trauma to the cranium.

In making the diagnosis roentgenographically, it is imperative to obtain well positioned stereoscopic lateral roentgenograms as well as basal or submentovertical views. Anteroposterior views may be more helpful in examining the temporal bone than posteroanterior projections.

Thirty-two roentgenograms; 3 photographs.

J. F. BERRY, M.D.
Louisville General Hospital

Carotid Angiography with Urokon, Using the Chamberlain Bi-plane Stereoscopic Angiographic Unit. Report of One Hundred Cases. Paul Lin, Frederick Murtagh, Henry Wycis, and Michael Scott. J. Neurosurg. **10**: 367–372, July 1953.

One hundred consecutive carotid angiograms were obtained with Urokon (30 per cent) in 72 patients.

The Chamberlain bi-plane stereoscopic angiographic unit was used, thus permitting stereoscopic films to be taken of the arterial and venous phases, with only 8 to 10 c.c. of contrast medium for the entire study. The authors briefly review the literature on contrast media for cerebral angiography and describe their technic.

There was only one complication, transitory unconsciousness, in this series of cases. The angiogram in this instance showed the arterial phase to be excessively prolonged, to 4.5 seconds, which may have been due to arterial spasm produced by the contrast medium. The patients experienced milder subjective complaints with Urokon 30 per cent than with Diodrast 35 per cent.

Six roentgenograms; 1 photograph.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Fourteen-Year Report of Facial Growth in Case of Complete Anodontia with Ectodermal Dysplasia. Bernard G. Sarnat, Allan G. Brodie, and W. Howard Kubacki. Am. J. Dis. Child. **86**: 162–169, August 1953.

A white boy twenty-one months of age was referred to the authors in 1938 because no teeth had appeared. Dental films had shown complete absence of teeth and tooth buds. The patient did not perspire. Physical examination revealed an alert boy with albinoid, lanugo-like hair sparsely distributed over the scalp. He had no eyebrows or eyelashes and no axillary hair. The skin of the lower eyelids was hyperpigmented and strikingly wrinkled toward the inner canthus. Lacrimation was observed.

When the patient was about six years of age, full lower and upper dentures were constructed, in order to improve mastication and facial appearance. Up to the age of sixteen, four additional sets had been required because of progressive growth of the jaws. At that age there was still complete anodontia, with ectodermal dysplasia manifested by lack of sweat and sebaceous glands. The sparse lanugo-like hair was increased in amount but lacked pigment.

Serial cephalometric roentgenograms were obtained in this case at six- to twelve-month intervals from the age of twenty-two months to sixteen years. These revealed the patient's overall measurements to be equal to small normal. A slightly less than normal nasion to anterior nasal spine dimension, with a markedly greater than normal vertical measurement within the nasal cavity proper, was noted. This appearance was found to be due to an over-expansion of the nasal cavity, which was shared by the paranasal sinuses. There was no "buckling" of the palate such as is typical of a variety of facial growth arrests. Further investigation is indicated to determine whether the over-expansion of the nasal cavity and sinuses is symptomatic of this type of dysplasia or traceable to a general weakening of the facial skeleton induced by lack of normal dental function.

The authors report briefly a second, similar case in a child first seen at twenty-six months.

Dental or lateral jaw roentgenograms will determine the absence of teeth and tooth buds in these cases and differentiate them from failure of eruption due to other causes.

Two roentgenograms; 7 photographs; 3 drawings.

C. R. PERRYMAN, M.D.
Pittsburgh, Penna.

THE CHEST

Agenesis of the Lung and Patent Ductus Arteriosus with Reversal of Flow. Report of a Case. Daniel S. Lukas, Charles T. Dotter, and Israel Steinberg. New England J. Med. **249**: 107–109, July 16, 1953.

Agenesis of the lung is rare. Diagnosis in the 57 cases previously reported was made at autopsy in all except 21. The increasing use of bronchoscopy, bronchography, and angiocardiography probably accounts for the more frequent antemortem recognition in recent years. The lesion occurs three times more frequently in the left than in the right lung, is seen more commonly in males, and is usually fatal early in life, as a result of a complicating infection or associated congenital anomalies. Marked shift of the mediastinum without apparent cause should suggest the diagnosis of pulmonary agenesis and lead to further investigation by special studies.

The authors report the case of a 22-year-old male with a history of repeated episodes of respiratory difficulty since birth. Clubbing and cyanosis of the toes had been present for the past eight years. Mediastinal shift to the left was apparent on physical examination. Also noted were inconstant murmurs in the left second intercostal space, the left axilla, and the interscapular region. Polycythemia and electrocardiographic changes were observed.

A plain chest roentgenogram showed displacement of the trachea and heart to the left. It was not possible to identify the great vessels and cardiac chambers in the homogeneous density that occupied the lower two-thirds of the left side of the chest. Bronchography demonstrated absence of the left main bronchus. Angiocardiograms established the cardiovascular nature of the structures in the left chest, with a greatly dilated main-stem pulmonary artery. The left pulmonary artery was absent, being replaced by a short patent ductus arteriosus, 16 mm. in diameter. The descending aorta and pulmonary artery became opacified simultaneously, indicating right-to-left flow through the ductus. Persistent opacification of the pulmonary artery during filling of the left ventricle and ascending aorta suggested a left-to-right ductal shunt. The bidirectional flow was confirmed by catheter studies.

Clubbing of the toes with cyanosis and normal appearing fingers reflect the bidirectional dynamics seen in severe pulmonary hypertension in association with a patent ductus arteriosus. This results as unoxygenated blood enters the descending aorta below the origin of the subclavian artery.

Four roentgenograms.

EDWARD E. TENNANT, M.D.
Jacksonville, N. C.

Mucocele of the Lung Due to Congenital Obstruction of a Segmental Bronchus: A Case Report; Relationship to Congenital Cystic Disease of the Lung and to Congenital Bronchiectasis. Beatty H. Ramsay. Dis. of Chest **24**: 96–103, July 1953.

Following a report of a mucocele of the lung in a fifteen-year-old girl, the author discusses the origin of mucoceles and their probable relationship to congenital cystic disease and bronchiectasis.

At operation in the case reported, a diaphragm-like obstruction was noted at the commencement of the involved bronchus. All the bronchi tributary to the

obstruction were dilated and filled with mucus. A thin septum of this nature has been noted in most bronchogenic cysts. These septa are probably congenital. The author believes that, if the specimen from his case had been examined by multiple section technic, the picture might have been one of multiple bronchogenic cysts, implying both a separation of the elements and multiple bronchial obstructions. It is suggested that perhaps most, if not all, so-called multiple intrapulmonary bronchial cysts are actually examples of bronchial mucoceles and that if, in the diseased portions of the lung, the bronchial systems were traced in a retrograde manner, the true situation would be recognized. The small outpouchings usually seen in the walls of bronchial cysts are almost certainly dilated bronchial branches partly taken up in and partly compressed by the major cyst. Thus it seems likely that such a cyst is merely an advanced stage of a pulmonary mucocele.

Congenital bronchiectasis is believed to be another varient of mucocele, occurring when the septum ruptures after dilatation of the tributary system has taken place.

A further finding of interest in this case was the normal inflation-deflation phenomenon of the involved segment, suggesting that the air passage was either transalveolar or transsegmental. This finding has been reproduced experimentally. It does not occur if there is parenchymatous disease of the occluded segment or if the main lobe bronchus is occluded.

Still another point of interest in the author's case was the failure of the mucus-filled tributary bronchi to produce significant shadows on the roentgenogram. This may have been due to an unfavorable density contrast as a result of scattering of the pathological process throughout distended lung of considerable depth.

A final finding was the absence of pigmentation of the affected areas, suggesting a filtering effect. Supporting this theory is the occurrence of unusually heavy deposits of carbon at the intersegmental planes bordering an obstructed segment.

A classification of pulmonary cysts is presented. Of the mechanical group, bronchogenic cysts arise from obstruction to a portion of the bronchial tree, while parenchymal cysts are the result of interference with collateral ventilation, with consequent over-distention. A similar interference with collateral ventilation, resulting in loss of air filling, may explain the areas of plate-like atelectasis frequently associated with minor inflammations and/or edema of the lower lobes.

[The case reported here appears also in J. Thoracic Surg. **26**: 21, 1953. Abst. in Radiology **62**: 608, 1954.—ED.]

Four illustrations, including 2 roentgenograms.

PAUL MASSIK, M.D.
Quincy, Mass.

Clinical and Theoretical Considerations of Plate-Like Atelectasis. R. Haubrich. Fortschr. a. d. Geb. d. Röntgenstrahlen **79**: 32–43, July 1953. (In German)

The author analyzed 140 cases of plate-like atelectasis, many of which were asymptomatic and were recognized only incidentally. Eighty-five cases were apparently the result of abdominal conditions and 55 of intrathoracic disease. The responsible abdominal lesions were found largely in the liver, gallbladder, and spleen, with a smaller number in the stomach and kidneys. The atelectasis characteristically occurred on the same side as the disease in the abdomen. The underlying pathological changes in the chest were chiefly cardiac; pneumonitis and acute infections were of less frequent occurrence. In many cases conditions involving both heart and abdomen were present. An associated bronchitis was a frequent finding. The atelectasis may be unilateral or bilateral and seems to have some predilection for the dorsal basilar segments of the lung, just above the level of the diaphragm. It is practically always easily identifiable roentgenologically and may be of relatively slight significance. It often disappears after correction of the underlying cause.

Various theories in explanation of this condition are discussed. Some authors believe that it is on the basis of a segmental reflex mechanism, with resulting active contraction of the affected layer of lung. Others consider it secondary to disturbance of diaphragmatic function. The latter view presupposes a mechanical basis, whereby restriction of diaphragmatic motion allows the collection of secretion in the finer bronchi, with resulting anoxia and atelectasis. The author prefers this explanation. The fact that phrenic paralysis with a high diaphragm is frequently seen in the absence of plate-like atelectasis is considered as evidence against the reflex theory.

In the early stage, the borders of the atelectatic area are not sharply demarcated and the reaction is reversible. In the later stages, there is sharper definition, with scar formation, and resolution is more gradual or fails to occur. Plate-like atelectasis is relatively seldom associated with pleural exudate, and in some cases no accompanying pathological process (pleuritis, etc.) is recognizable. Kymography demonstrates increased costal motion and decreased diaphragmatic motion in the involved region.

[Although the above description is largely theoretical, it indicates that we would do well to search carefully for associated pathology whenever plate-like atelectasis is recognized.—E. W. S.]

Six roentgenograms; 1 drawing.

E. W. SPACKMAN, M.D.
Fort Worth, Texas

Tuberculous Bronchitis and Bronchiectasis. G. Simon. J. Fac. Radiologists **5**: 33–41, July 1953.

The radiological appearance in cases of endobronchial tuberculosis is correlated with pathological findings. The author divides the lesions into those of the proximal bronchi (within the range of vision of the bronchoscopist) and those of the more peripheral bronchi.

An endobronchial tuberculous lesion of one of the more proximal bronchi frequently results in a slowly progressive narrowing, with consequent obstructive atelectasis and its associated changes (alveolar edema, parenchymal inflammation, or bronchiectasis). Roentgenograms will show the homogeneous shadow of the atelectatic lobe, and bronchoscopy will often confirm the cause. Occasionally the shadow of the lobe is not homogeneous but contains circular translucent areas which may be mistaken for cavities in a fibroid lung. A relatively uncommon effect of the endobronchial lesion is a partial obstructive emphysema. In some cases bronchiectasis and consolidation occur, rather than collapse, producing a pyramidal, not quite homogeneous density. Bronchography and tomography

may be necessary to demonstrate the type of change present.

Lesions beyond the vision of the bronchoscopist may be difficult or impossible to diagnose preoperatively. An endobronchial abscess may produce a homogeneous circular shadow, occasionally with smaller rounded densities grouped about it. These circular shadows, when they exceed 1.0 cm. in diameter, should be further investigated by tomography if active tuberculosis is suspected. This type of disease tends to resist all forms of therapy except resection. A dilated thick-walled bronchus may simulate an ordinary parenchymal cavity. The so-called "tennis racket" appearance is produced by a distended bronchus with a tubular shadow (thickened bronchus) running from it toward the hilus. Distended bronchi containing caseous material may produce elongated finger-like opacities. This last appearance, however, may be simulated by actual parenchymal foci surrounded by a thick wall of fibrous tissue.

The author presents numerous cases demonstrating tuberculous endobronchial disease in its varied manifestations. The tomograms, bronchograms, and photographs of the resected specimens afford an interesting radiographic-pathologic correlation.

Fourteen roentgenograms; 6 photographs.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Coin Lesions of the Lung. Clifford F. Storey, Roald A. Grant, and Bruce F. Rothmann. Surg., Gynec. & Obst. **97:** 95–104, July 1953.

The importance of small solitary circumscribed pulmonary lesions which give rise to no symptoms has only recently begun to be appreciated. The term "coin lesions" has been applied to these nodules because they appear round in the postero-anterior chest roentgenogram. In reality they are spheroid rather than disk-shaped. The authors propose to limit the term to solitary lesions 1 to 5 cm. in diameter, round or oval in shape, sharply circumscribed and surrounded on all sides by normal lung tissue, homogeneous in density or containing calcium, and asymptomatic.

The incidence of cancer in reported series of such lesions has varied widely—from 7.5 to 55 per cent—due to lack of a precise definition for the term coin lesion and the selectivity of the material. Because so many of the lesions have proved to be malignant, most authors advocate prompt excision.

Observations on 40 consecutive patients with coin lesions histologically verified are here reported. Most of the group were apparently healthy young males on active duty in the Armed Services. In each instance the pulmonary lesion was discovered by chance, on a routine chest film. The incidence of cancer was 17.5 per cent. Seventy per cent of the cases were classed as "tuberculomas," more frequently reported as "chronic fibrocaseous pulmonary granuloma compatible with a tuberculous origin." Ten per cent of the tuberculomas were actually blocked tuberculosis cavities. The remaining lesions represented various benign conditions.

Five case histories are presented. In one case a lesion in the right upper lobe (in a 24-year-old male) remained stable for nineteen months. Time-consuming diagnostic studies were undertaken, and during the interval widespread metastases occurred. Death ensued from alveolar-cell carcinoma.

In a high percentage of cases, an accurate diagnosis can be made only by the pathologist. The authors state that the overwhelming majority of diagnostic endeavors will prove to be fruitless. The lesion should be localized with postero-anterior and lateral chest roentgenograms. Tomograms usually do not offer any vital information not provided by standard films. Bronchoscopy should be done routinely, especially to rule out a mucosal lesion involving the larger bronchi and to obtain material for bacteriologic and cytologic studies. Skin tests for tuberculosis, histoplasmosis, and coccidioidomycosis are carried out in all patients, but are actually of no practical value in the individual case. Since these patients had no symptoms or significant physical findings, the authors did not think it practical to do barium enema studies, gastrointestinal series, etc., to rule out possible metastatic lesions.

A fairly even distribution among the five lobes is observed, so that it may be said that coin lesions have no characteristic location.

The authors are convinced that surgical excision without undue delay is the treatment of choice. Operation was done in 38 of their 40 cases, with no surgical mortality. Operative procedures consisted of lobectomy in 3 instances, segmental resection in 2, wedge resection in 6, local enucleation in 26, wedge resection and lobectomy in 1. In all cases the mass is sectioned at the table and frozen section performed if necessary.

Seven roentgenograms; 2 tables.

JAMES LYON, M.D.
University of Pennsylvania

Carcinoma of the Middle-Lobe Bronchus. G. Brian Locke. J. Fac. Radiologists **5:** 1–18, July 1953.

Carcinoma of the middle-lobe bronchus is uncommon, having been reported in from 0.6 to 11 per cent of patients with primary lung cancer. The author reports his experiences with 20 cases, reviews the literature on the subject, and discusses at some length the anatomy of the middle lobe with reference to collapse and consolidation.

Radiological study of the middle lobe is accomplished with conventional postero-anterior and right lateral projections, which may be supplemented by a lordotic view. A summation shadow is sometimes seen in the lateral view, representing a high right diaphragm and the heart. This may simulate a middle-lobe consolidation. Tomography and bronchography are of great assistance. Tomography in the lateral position will show accurately the shape and location of the lesion. The right posterior oblique position, however, is more helpful and may show the whole of the bronchus on one film. On the bronchogram, an abrupt straight-across "stop" in a bronchus of normal caliber favors tumor, whereas a smooth tapering narrowing is more likely to be due to a fibrous stricture or obstruction by an extrinsic lesion. Fluoroscopic examination with a barium swallow should be included to determine the presence of pericardial fluid and mediastinal node enlargement.

In the radiologic differential diagnosis of right middle-lobe carcinoma, a great variety of lesions must be considered. Encysted interlobar effusion or empyema has attracted much attention. The precise location of the opacity should be determined by tomography, and bronchographic confirmation may be desirable. Other rarer extrapulmonary conditions in this region are pericardial cyst, dermoid, and diaphragmatic hernia.

The main group of lesions to be considered are those causing consolidation with or without collapse. Inflammatory consolidation of the middle lobe alone is rare, but it does occur. An abscess may accompany an abscess of the apex of the right lower lobe; it is uncommon as an isolated finding. A solitary pulmonary metastasis was seen in 2 cases of this series. The "middle-lobe syndrome," or post-tuberculous bronchiectasis and bronchostenosis of the middle lobe, is due to hilar node enlargement obstructing the bronchus.

Bronchial carcinomas in the middle lobe may be divided into two groups: the rather rounded peripheral tumor and the central tumor causing lobar or segmental collapse. In this series, the central type was more common. Extension up the pulmonary vein to involve the pericardium is frequent. Pleural effusion in any significant quantity and phrenic paralysis were not seen on initial examination. There were metastases to hilar lymph nodes, bone, and brain. Some cases were followed over a period of many months without alteration in the radiological appearance. It is thought that the interlobar septa act as resistant confining barriers to the growth. Hemoptysis was the commonest presenting complaint, chest pain and loss of weight also being frequent symptoms. Since the middle-lobe bronchus is the least accessible to bronchoscopic examination, this method of study was disappointing.

The author presents 14 cases demonstrating benign and malignant diseases of the middle lobe and the difficulties attendant upon diagnosis.

Thirty roentgenograms; 3 diagrams; 1 photograph.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Idiopathic Juvenile Pulmonary Haemosiderosis. C. J. Hodson, N. E. France, and I. Gordon. J. Fac. Radiologists **5:** 50–61, July 1953.

The radiographic appearances of the lungs in cases of idiopathic juvenile pulmonary hemosiderosis are quite remarkable and in many instances characteristic. The authors report 3 cases and describe the pulmonary changes in detail.

The main pathological features appear to result from widespread hemorrhage into the lung tissue, either as a steady seepage or as multiple local hemorrhages. Innumerable phagocytes containing coarse granules of hemosiderin are scattered through the alveoli, interstitial tissue, and lymphatics. Superimposed on this general picture are small focal hemorrhages. In later stages there is degeneration of elastin in the alveolar walls and in the media of the small vessels. Accompanying these changes is an increase of reticulin. Occasionally the right heart may show hypertrophy or the liver and spleen may be enlarged.

The age of onset is usually under ten years. Two main clinical types are seen. One is characterized by recurrent acute episodes, ending fatally in a matter of months or gradually producing a chronic effect; in the other, the disease is chronic throughout. The acute episodes usually present as an upper respiratory infection with fever, dyspnea, cough, cyanosis, and lethargy. There may be bouts of chest or abdominal pain. Pallor develops rapidly. Hemoptysis or hematemesis invariably appears, becoming a constant part of the picture. The physical signs are variable and inconstant. The blood shows a severe hypochromia and occasionally evidence of hemolysis; the bone marrow a normoblastic reaction. The anemia in the early stages responds well to iron therapy and the child is in apparent good health in the intervals between acute attacks.

The radiographic appearances seem to be of two main patterns, which are often superimposed. One consists of multiple, "acute looking" blotchy shadows scattered irregularly throughout both lung fields. The lesions vary in size from a few millimeters to 2 or 3 cm. in diameter, and the distribution changes over short periods of time. The other appearance is that of a slowly developing but persistent change of a "reticular" type. It involves mainly the middle and lower zones of the lung fields and gradually increases in density.

Severe symptoms may occur without radiographic changes, or the reverse may be true. More often the acute episodes are accompanied by findings in the chest of the "blotchy" type and, in the later stages, by the "reticular" pattern. Either picture may predominate.

In an attempt to correlate the radiographic findings with the pathological changes, the authors radiographed thin slices of lung obtained at autopsy. They found that the "acute blotchy" type of shadowing was caused by multiple small hemorrhages and not by collapse, as was previously thought. The striate pattern was due to the deposition of hemosiderin and siderophages in the septa and lymphatics of the lungs.

The fundamental lesion which permits the escape of blood cells from the circulation is not known, nor is it certain that the disease is always fatal.

Ten roentgenograms; 2 photographs.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

A Case of Moniliasis of the Lungs. J. Smulewicz. Schweiz. med. Wchnschr. **83:** 702–703, July 25, 1953. (In German)

Except in certain tropical and subtropical regions, primary fungous disease of the lungs has been uncommon. Recently reports have begun to appear of both probable and proved cases of pulmonary fungous infection which either followed or were associated with intensive antibiotic therapy.

A case is reported of a woman of 34 years in whom bizarre parenchymal lung changes appeared on the roentgenogram during the course of intensive penicillin treatment for what appeared to be an ordinary bronchopneumonia. Bilateral, poorly defined, irregular infiltrates, with the denser lesions toward the bases, were noted, and several cavities with fluid levels developed. The entire process partially resolved during a six weeks period and was entirely gone in seven months. During the first week a sputum culture yielded penicillin-sensitive streptococci and staphylococci. Following this, and during the height of the lung changes, pure cultures of *Monilia* (*Candida*) *albicans* could be grown.

Four roentgenograms.

WILLIAM F. WANGNER, M.D.
Royal Oak, Mich.

Respiratory Disorders Among Welders. Robert Charr. J.A.M.A. **152:** 1520–1522, Aug. 15, 1953.

Long continued exposure to arc welding fumes may produce exaggerated linear markings and nodulations in the lungs which are indistinguishable roentgenographically from silicosis. However, if exposure is terminated there may be a regression of these changes.

Ordinarily the accompanying pulmonary symptoms are not incapacitating, but occasionally a change of occupation is necessary. There does not appear to be any increased incidence of tuberculosis among welders.

The author presents 7 cases of respiratory disorders in welders. In 3 the symptoms were severe enough to necessitate a change of occupation.

Three roentgenograms. JOHN J. CRAVEN, M.D.
Cleveland Clinic

Arterio-Venous Aneurysma of the Lung. Jan Muri. Dis. of Chest **24:** 49–61, July 1953.

The author describes and illustrates a case of arteriovenous aneurysm of the lung and presents a general discussion of the subject, listing the 79 cases found in the literature with their clinical features and outcome. Cyanosis, clubbing, and secondary polycythemia with normal heart sounds and a bruit over one segment of the lungs are suggestive findings. Conventional roentgenograms and angiography confirm the diagnosis. The latter are doubly valuable, since multiple aneurysms are common. Lindgren has given an excellent account of the roentgen findings (Acta radiol. **27:** 585, 1946. Abst. in Radiology **50:** 262, 1948).

The complications of arteriovenous aneurysm are rupture of the aneurysm into a bronchus or pleural space, infection, especially brain abscess, and neurological symptoms. The treatment is surgical removal.

Four roentgenograms; 2 photographs; 1 table.

PAUL MASSIK, M.D.
Quincy, Mass.

Pulmonary Manifestations of Scleroderma. Wade H. Shuford, William B. Seaman, and Alfred Goldman. Arch. Int. Med. **92:** 85–97, July 1953.

This report is based on a study of 37 patients for whom a clinical diagnosis of scleroderma was established. Five patients had abnormal chest roentgenograms. In 4 of these the clinical diagnosis was supported by histologic evidence. The roentgen findings in this group were strikingly similar, consisting of a diffuse, linear, interstitial infiltration, more marked in the lower half of both lung fields. In a sixth case, in which initial chest roentgenograms showed no abnormalities, a terminal pulmonary edema developed. Scleroderma of the lung may clinically precede the development of cutaneous sclerosis and should be considered in the differential diagnosis of obscure cases of pulmonary fibrosis.

Pulmonary manifestations of other collagen diseases are discussed. When the roentgenographic appearance is correlated with the clinical findings, a more precise diagnosis may be made.

Nine roentgenograms.

THEODORE E. KEATS, M.D.
University of California

Bronchial Obstruction with Lobar Atelectasis and Emphysema in Cystic Fibrosis of the Pancreas. Paul A. di Sant'Agnese. Pediatrics **12:** 178–190, August 1953.

In 22 of 211 infants (10.4 per cent) with cystic fibrosis of the pancreas seen in a twelve-year period, atelectasis of one or more pulmonary lobes was noted during the period of initial pulmonary involvement. Atelectasis was a terminal complication in 2 additional cases not included in this study. Respiratory symptoms were noted before three months of age in 15 cases, before six months in 21. Lobar atelectasis was also recognized early, in 18 cases before six months of age.

Consistency of distribution of the atelectasis was striking. In no case was the left lung involved. The right upper lobe was the most frequent site, followed by the right middle lobe. The lower lobe was never involved alone. In all cases but one, emphysema was present on admission or developed subsequently. With one exception, the emphysema affected all portions of the uncollapsed lung.

Fifteen of the 22 patients died. The therapeutic value of antibiotics was demonstrated by the fact that all of the 7 survivors were born after the advent of penicillin. Bronchoscopy was ineffective as a therapeutic measure.

During the same period (twelve years), 48 cases of lobar atelectasis were found in infants under one year of age. Ten had proved cystic fibrosis of the pancreas, while 10 others had a clinical picture consistent with congenital pancreatic deficiency, though the diagnosis was unconfirmed. The remainder had congenital heart disease, vascular anomalies, etc. Congenital atelectasis was excluded from this series. In this group, only one possible fibrocystic patient had involvement of the left lung.

The basic cause for pulmonary involvement in fibrocystic disease of the pancreas remains unsettled. It may be attributed (1) to a breakdown of normal mechanisms for removal of bronchial secretions, due to failure of intestinal absorption of some unknown substance, or (2) to the occurrence of abnormal bronchial secretions which are difficult to remove. Whatever the difficulty, bronchial obstruction ensues.

It is postulated that, because of retained bronchial secretions, acute respiratory infections lead to increased mucus production. Widespread bronchial obstruction ensues and the infection becomes widespread. Repeated cycles of similar episodes lead eventually to death. The author views obstruction as the primary manifestation, complicated secondarily by infection.

Six roentgenograms; 1 graph; 3 tables.

J. MAURICE SWAIN, M.D.
Cleveland Clinic

The Size of the Heart Chambers Determined by Cardiac Catheterization. A. Schaede and P. Thurn. Fortschr. a. d. Geb. d. Röntgenstrahlen **79:** 21–32, July 1953. (In German)

By conventional x-ray studies it is frequently difficult or impossible to determine accurately the size of the individual heart chambers. This information, however, particularly as regards the ventricles, is often of special value in differential diagnosis. For example, the relative size of the ventricles may in certain instances be the determining factor in making a distinction between atrial septal defect and patent ductus.

In heart catheterization studies, certain anatomical factors must be considered in the interpretation of the roentgenographic findings. To confirm right ventricular enlargement, the catheter must lie against the septum. Entrance into the coronary sinus may give a totally false impression of enlarged right ventricle. A left anterior oblique projection, at an angle of 60 degrees, aids in visualization of the right ventricle. Enlargement of this chamber is indicated when the

catheter shows a convex curve toward the spine (dorsally). Appearance of a large portion of the heart shadow dorsal to the catheter and above the diaphragm indicates enlargement of the left ventricle. These signs, taken into consideration with the pressure changes and relative oxygen saturation, may clear up an otherwise obscure case which could easily be misinterpreted in the usual radiographic studies.

The author quotes an example in which a mistaken diagnosis of combined mitral lesion might easily have been made from the conventional x-ray studies. The fact that the left cardiac border projected posteriorly would appear to indicate enlargement of the left ventricle. On catheterization, however, it was shown that the prominent left border was caused by dilatation of the right ventricle and that the lesion was a pure mitral stenosis.

Other contributory evidence should never be overlooked. The study of the barium-filled esophagus in demonstrating left auricular enlargement is essential. A large left auricle may depress the outflow tract of the right ventricle. A cathether may identify congenital cardiac conditions by its unusual position in passing through false openings, etc. An enlarged right ventricular outflow tract should always be recognized. All of these factors considered together in relation to the clinical and physical aspects of the case are necessary in arriving at a final diagnosis.

Twenty-three roentgenograms.

E. W. SPACKMAN, M.D.
Fort Worth, Texas

Contribution to the Topography of the Cardiac Orifices and Their Interrelationship. Opacification of the Coronary Vessels. Georges Apostolokis and Issidore G. Gounaris. J. de radiol. et d'électrol. **34:** 516–518, 1953. (In French)

A method is described for the study of the anatomic relations of the cardiac valves and coronary vessels at autopsy. After the pericardium is incised, metallic rings are placed along each valve and roentgenograms are taken in various projections. To study the coronary vessels, the coronary orifices are injected with a metallic preparation. These studies may help determine the location of various valves and vessels when calcification is seen during fluoroscopy or radiography.

The text is best read with the reproduced radiographs present for inspection. An attempt will be made in this abstract to give a brief description of the figures showing the valves. In all projections the tricuspid valve is lowest and the pulmonic valve is highest in position. Also, the mitral and pulmonic valves show a greater inclination from the horizontal, so that a more nearly circular projection is shown.

In the postero-anterior projection, the tricuspid valve is found in the right third of the cardiac silhouette, in the region of the right cardiophrenic angle. The mitral valve is located in the left half of the cardiac silhouette, over the left vertebral border, medial to the left auriculoventricular region. The aortic and pulmonic valves are in the middle third, superiorly, overlying the vertebral shadow, with the pulmonic valve slightly higher and to the left, and presenting a greater inclination from the horizontal.

In the left anterior oblique view, the tricuspid valve is seen low in the anterior third of the cardiac silhouette. The mitral valve is at a slightly higher level in the posterior third, and the aortic and pulmonic valves are located superiorly in the middle third. Again the pulmonic valve is slightly higher and slightly more inclined.

In the right anterior oblique view, the tricuspid valve is low in the posterior half of the cardiac silhouette. The mitral valve is located slightly higher in the middle third and the aortic valve is seen superiorly in the middle third. The pulmonic valve is seen in the anterior third of the silhouette, slightly superior to the level of the aortic valve.

Four roentgenograms.

CHARLES NICE, M.D.
University of Minnesota

Heart Disease Discovered on Chest Microfilms. R. V. Slattery. J.A.M.A. **152:** 1595–1596, Aug. 22, 1953.

To determine the accuracy of microfilm chest surveying in detecting heart disease, 70-mm. films and clinical findings of 682 patients admitted to the outpatient departments of the Chicago Clinics were compared. Clinical evidence of heart disease was found in 56, of whom 25 had abnormal cardiac silhouettes demonstrated on miniature films. In 12 patients abnormal silhouettes were found without clinical confirmation of heart disease. The types of cardiac involvement detected and missed were as follows:

Type of Heart Disease	Clinical Heart Disease, No.	Detected on Microfilms No.	Detected on Microfilms %	Not Detected on Microfilms No.	Not Detected on Microfilms %
Rheumatic	7	4	57.1	3	42.9
Hypertensive	21	11	52.4	10	47.6
Coronary	11	2	18.2	9	81.8
Arteriosclerotic	14	6	42.9	8	57.1
Syphilitic	2	1	50.0	1	50.0
Congenital	1	1	100.0	0	0
Total	56	25	44.6	31	55.4

In view of the low degree of detection—45 per cent—and the fact that 90 per cent of the patients had generally recognized cardiac symptoms, the author concludes that the use of miniature survey filming for the detection of heart disease is not to be recommended.

Two tables.

FRED J. HODGES, III, M.D.
University of Michigan

Primary Myocardial Disease in Infancy and Childhood. Harold D. Rosenbaum, Alexander S. Nadas, and Edward B. D. Neuhauser. Am. J. Dis. Child. **86:** 28–44, July 1953.

Forty-five patients were seen at the Children's Medical Center (Boston) with a syndrome of myocardial disease characterized by cardiomegaly, absence of significant murmurs, electrocardiographic abnormalities, and normal blood pressure. Pathological observations in 26 cases coming to autopsy are analyzed.

The autopsied cases are classified into two groups. In the first were 6 patients having rare conditions which are seen usually in infants under six months of age and in which congestive heart failure is uncommon. The diagnoses were glycogen-storage disease of the heart (3 patients), aberrant left coronary artery arising from

the pulmonary artery (1 patient), and medial necrosis of the coronary arteries (2 patients).

The remaining 20 cases were divided equally between idiopathic myocarditis of Fiedler and subendocardial sclerosis. In these conditions the age of onset is frequently beyond six months, and congestive failure is usually present, often severe. In contrast to the first group, these patients were found to be amenable to treatment responding favorably to digitalis. In approximately half of the group the T-wave changes and left ventricular hypertrophy disappeared or became less marked. In about an equal number of cases, the heart became smaller, occasionally reaching normal size.

Roentgen findings were similar in the cases of both groups. The enlargement of the heart was massive and generalized, involving all chambers. Usually the cardiac silhouette was globular, and it was not unusual for the heart to be in contact with the left lateral chest wall. Except for engorgement in some cases, the pulmonary vessels were not remarkable. Absence of x-ray evidence of increased or decreased pulmonary blood flow is valuable in excluding left-to-right shunts and pulmonic stenosis, respectively. Pulmonary congestion was present in 18 of a total of 35 patients in whom this factor could be evaluated.

A family history of the disease and a skeletal muscle biopsy showing high glycogen content may be valuable, the authors believe, in detecting glycogen-storage disease of the heart. The appearance of symptoms in "attacks" and the roentgen demonstration of a bulging prominence in the region of the left ventricle, due to thinning of the myocardium in the area, will occasionally permit the diagnosis of an aberrant left coronary artery. Onset of symptoms before three months of age, associated congenital anomalies, and evidence of a systemic disease suggest the presence of medial necrosis of the coronary arteries. Idiopathic myocarditis and subendocardial sclerosis without vascular involvement are indistinguishable clinically. For this reason, the exact diagnosis in the 19 patients who survived was not ascertained, but myocarditis is considered likely.

Eight roentgenograms; 3 electrocardiograms; 5 tables.

ARTHUR S. TUCKER, M.D.
Cleveland Clinic

Endocardial Fibroelastosis. Report of Unusual Case with Impaired Ability to Fabricate Serum Proteins. Ben E. Katz and Forrest H. Adams. Am. J. Dis. Child. **86:** 186–197, August 1953.

A case of endocardial fibroelastosis in a six-year-old child is reported, with clinical and laboratory findings and postmortem observations. An unusual feature was the occurrence of persistent edema, believed to be due in part to low plasma proteins secondary to impaired ability of the liver to fabricate such proteins. Roentgen examination revealed diffuse enlargement of the heart, with a prominent left auricle and a convex pulmonary artery segment.

The condition was not diagnosed during the patient's life, though the possibility of fibroelastosis was at one time considered. [In a recent article in Radiology (**61:** 865, 1953) Golper describes a case of endocardial fibroelastosis, with special emphasis upon the radiologic aspects.]

Three roentgenograms; 2 electrocardiograms; 7 photographs and photomicrographs; 1 graph.

THE DIGESTIVE SYSTEM

The Survey Roentgenograms as an Aid in the Diagnosis of Acute Abdominal Conditions. W. Wayne Sands. Surg., Gynec. & Obst. **97:** 4–10, July 1953.

Survey films of the abdomen coupled with proper interpretation by a roentgenologist are a valuable aid in the diagnosis of abdominal emergencies. The author not only discusses the technic and the various abdominal conditions which may be diagnosed roentgenographically, but also illustrates the anatomical landmarks as seen on the film and describes the manner in which materials of varying density are contrasted.

A complete roentgen examination in the presence of an acute abdominal condition should routinely include at least four films: a high and low supine film, an upright film, and a postero-anterior chest film. A technic with a wide latitude of contrast is preferable to aid in distinguishing the anatomical landmarks, since the patient is usually not prepared by enemas or cathartics and a contrast medium is not used.

Few things plague the radiologist so persistently as the interpretation of distorted and displaced bowel segments. In the differentiation of bowel obstructions the sins of omission are particularly apt to haunt him. The more common abdominal conditions are ileus, mechanical bowel obstruction, and peritonitis. Also discussed are congenital bowel obstruction, mesenteric vascular occlusion, volvulus, gallstone obstruction, intussusception, aortic aneurysms, pancreatic disease, and ruptured spleen.

The postero-anterior chest film is a necessary part of the examination because it is well known that thoracic diseases may cause abdominal pain. Pneumonia and acute pneumothorax may commonly simulate abdominal emergencies; other thoracic conditions which may mimic acute abdominal disease are dissecting aortic aneurysm, ruptured esophagus, acute pericarditis, Pott's abscess, osteoarthritis, and vertebral fractures.

Eight roentgenograms.

HENRY P. PENDERGRASS, M.D.
University of Pennsylvania

X-Ray Determination of the Decomposition of Fat and Its Fate in the Gastro-intestinal Tract. H. Lidl. Fortschr. a. d. Geb. d. Röntgenstrahlen **79:** 70–79, July 1953. (In German)

The anatomy and general motor function of the gastrointestinal tract have been observed extensively in the conventional type of x-ray examination, but the physiological factors have not been followed to the same degree. The present paper is concerned with the physiology of fat digestion, especially in the small bowel.

The following steps are important in fat digestion: (*a*) emulsification, (*b*) splitting into glycerin and fatty acids, (*c*) production of the water-soluble components. Normally there is no significant degree of fat digestion in the stomach. This takes place in the alkaline medium of the small intestine through the action of the fat-splitting enzyme (especially lipase). Milk fat is emulsified in the stomach, but other fats are digested in the stomach only when there is marked regurgitation through the pylorus. The chief fat-splitting ferment is the pancreatic lipase, activated by the bile. Lipase from the small bowel acts to a lesser degree. Since non-absorption of fat hinders the protein-splitting fer-

ment and influences calcium metabolism and the vegetative and hormonal systems, a simple practical method of demonstrating abnormal physiology in the digestion and absorption of fats is of great practical value.

One-half gram barium sulfate tablets were coated with a vegetable fat having a melting point of 41 to 43° C. In the normal gastrointestinal tract, these tablets should disintegrate in about three hours. If they are observed to be intact after four hours, delayed fat digestion is indicated. If they are intact in the colon, marked physiological changes are present. Normally, the tablet usually began to show dissolution in the lower jejunum, independent of any delay in the stomach. In some cases, dissolution did not occur until the lower ileum was reached. It is thus apparent that the activity the tract generally influences the location in which the tablets disintegrate, but gastric secretion appears to play relatively little part. In cases in which there had been resection of the stomach, there was increased rapidity of passage through the small bowel. After the Billroth II operation, intact tablets were frequently observed in the colon. After the Billroth I procedure, there appeared to be slower passage in the small bowel, with normial dissolution time.

This test is used also as an indirect estimate of gallbladder function. In the presence of a non-functioning gallbladder, dissolution time was prolonged. In cases with jaundice, tablets were regularly found intact in the colon. Removal of the gallbladder appeared to have no influence on the disintegration time.

Fourteen roentgenograms

E. W. SPACKMAN, M.D.
Forth Worth, Texas

The Proof of Esophageal Varices and Their Clinical Significance in Portal Hypertension. François Robert and Theo Hoffmann. Fortschr. a. d. Geb. d. Röntgenstrahlen **79**: 51–70, July 1953. (In German)

Portal hypertension may develop as a result of venous stasis produced by either extrahepatic or intrahepatic obstruction due to such conditions as thrombosis of the portal or splenic vein, congenital stenosis of the portal vein, cirrhosis, thrombosis of the hepatic vein, Chiari syndrome, etc. With the mechanical obstruction, back pressure develops in the portal system, with dilatation of the spleen and venous bed, and eventual overfilling of the esophageal plexus.

The submucosal plexus within the esophagus is loosely held in a network of fibrous tissue, and varicosities are easily formed, impinging on the lumen. Diagnosis may be made by x-ray examination and endoscopy. The latter, however, is considered dangerous in cases with recent hemorrhage or large varicosities, and in the very early stages is unreliable.

A carboxymethyl cellulose barium preparation is used. This adheres to the mucosa and shows very fine changes in the relief pattern. A spoonful of the mixture is given and, after an interval of two to five minutes, fluoroscopy and film studies are done, especially in the oblique position. A swallow of water is then given and further studies are made. Films are made at 8/100 of a second, 80 cm. distance, and 95 kv.

The very earliest stage is the result of diffuse venous filling of the submucosal plexus in the lower third of the esophagus. Slight irregularity and separation of the folds are demonstrable radiologically. At this stage, the evidence is considered merely suggestive. Later, dilatation of the individual veins is recognized, with elevation of the mucosa and protrusion into the lumen. As the condition develops, definite worm-like defects and nodular protrusions are recognized in the barium column, with involvement of the middle and upper thirds of the esophagus in very advanced stages. Irregular dilatation of the lumen is associated. As a rule there is only slight delay, if any, in passage of the barium, but esophageal residue of one hour duration has been observed. Twenty-two cases are briefly summarized in table form, with details of etiology and clinical aspects.

The size of the varices and the tendency to bleeding depend upon other factors than the degree of portal hypertension present (collateral circulation, condition of esophageal musculature, mucosal changes, etc.).

The following conditions are to be considered in differential diagnosis: (*a*) air bubbles, which are inconstant in various positions and on repeated studies, (*b*) impairment of the mucosal folds in elderly people, frequently disappearing on full inspiration, (*c*) curling or small adhesions, differentiated by the x-ray appearance and the history of associated conditions such as anemia, esophagitis, etc., (*d*) carcinomatous infiltrations, which are more sharply demarcated, causing rigidity and dysphagia.

Seventeen roentgenograms.

E. W. SPACKMAN, M.D.
Forth Worth, Texas

Volvulus of the Stomach. John P. West and Vann T. Floyd. Am. J. Surg. **86**: 112–115, July 1953.

Volvulus of the stomach which obstructs the esophageal and duodenal orifices is rapidly fatal if not promptly corrected. The usual case is due to incarceration and torsion of the stomach within a diaphragmatic hernia. Symptoms are those of acute high intestinal obstruction, without distention. Physical signs and x-ray examination usually indicate fluid and air in the chest. A less frequent type of volvulus occurs intra-abdominally, giving rise to severe pain, some distention, and constant nausea with unsuccessful attempts at vomiting. Roentgen examination shows a distended abdominal viscus, and a barium swallow demonstrates lower esophageal obstruction.

The authors present a case with typical physical and x-ray findings. The patient had refused surgery for a large hiatus hernia containing most of the stomach and apparently causing mild symptoms, six years previously. On admission, x-ray films revealed air-fluid levels presumed to be in the stomach, only a small part of which was above the diaphragm. Exploration disclosed an enormously dilated stomach rotated so that the pylorus was under the left diaphragm and the fundus was on the right with the major portion of the posterior wall presenting anteriorly. The stomach was emptied by trocar and replaced in its normal position. The patient did well, and two months postoperatively the stomach was found to have returned to the thorax in the hernia. Later the hernia was repaired and the stomach, though showing some slight persistence of abnormal rotation, did not again become herniated.

Three roentgenograms; 2 drawings.

GEORGE A. SHIPMAN, M.D.
New Orleans, La.

Strangulated Hiatus Hernia. G. A. P. Hurley. Ann. Surg. **138**: 262–267, August 1953.

Until recently, the opinion was widely held that her-

niation of the stomach through the esophageal hiatus of the diaphragm was a relatively benign condition, which could be easily managed medically. Two case histories are given to supplement other reports of recent years which leave no doubt that hiatus hernia, particularly when progressive or large in size, is an urgent indication for operation.

The first patient was an 18-year-old male who had a strangulated hernia of the stomach following trauma. Gangrene of the greater curvature occurred where the hiatus had gripped the stomach. The upper pole of the spleen was infarcted.

The second case was that of a 73-year-old woman with a strangulated hiatal hernia, the strangulated portion being tense and globular due to a tight constriction of the stomach by the hiatus. Surgical treatment was undertaken successfully in spite of the patient's age and poor medical condition.

There is little in the literature of diaphragmatic hernia with reference to strangulation. The few writers who have mentioned it emphasize that traumatic diaphragmatic hernia is particularly liable to this complication. The author thinks that strangulation of the stomach, with or without gangrene, is more common than the literature would suggest, but that owing to late diagnosis, or inability to appreciate the seriousness of the condition, many patients die without operation, or when diagnosis is made too late to be life-saving. Many may not even be admitted to hospitals.

The possibility of strangulation should be considered when (*a*) a large amount of stomach is incarcerated in the hernia; (*b*) the hiatus hernia first appears, or shows an increase in size, at the time of or shortly after a crushing injury of the abdomen or lower chest; (*c*) the stomach has undergone torsion or inversion, and the greater curvature lies high in the chest. The author emphasizes the diagnostic features and the importance of prompt surgical treatment by transpleural approach.

Six roentgenograms. PHYLLIS A. CROZIER, M.D.
Pittsburgh, Penna.

Stenosis of the Stomach Caused by Corrosive Gastritis. Alberto L. C. Maggi and M. Meeroff. Gastroenterology **24**: 573–578, August 1953.

A stenosis of the lower body and antrum of the stomach followed ingestion of a large quantity of hydrochloric acid by a 30-year-old female. The patient was seen, in Buenos Aires, about one year following the suicidal attempt, complaining of early postprandial vomiting with considerable effect on her general condition. There was no dysphagia.

Roentgenograms revealed a stricture starting abruptly at the junction of the middle and inferior thirds of the stomach and extending distally to involve the pyloric antrum. There was some dilatation above the stenosis. The esophagus was normal. Gastric analysis revealed a histamine-resistant achlorhydria. Gastroscopy showed an extensive superficial gastritis. Gastrectomy of the Reichel-Polya type was performed, and postoperatively the patient was perfectly well.

The significant features of the case are the sparing of the esophagus, which is seen only when the ingested caustic is an acid, and the early postprandial vomiting, in contrast to pyloric syndromes of cancer or ulcer, where the vomiting is usually late.

Nine roentgenograms; 2 photomicrographs.
JOHN F. RIESSER, M.D.
Springfield, Ohio

Coexistent Carcinoma of the Stomach and Hypertrophic Gastritis. Report of a Case with Review of the Literature. E. C. Texter, Jr., C. W. Legerton, Jr., R. J. Reeves, A. G. Smith, and J. M. Ruffin. Gastroenterology **24**: 579–586, August 1953.

Giant hypertrophic gastritis is a rare pathological condition characterized by large gastric mucosal folds which, on gastroscopic examination, appear tufted and wavy. The mucosal hypertrophy is associated with increase in glandular tissue and preservation of the membrana propria. Roentgenologically there is a gross alteration of the mucosal pattern to such an extent that the detection of a superimposed malignant tumor is extremely difficult. Rugae are very prominent and tortuous.

A relation between hypertrophic gastritis and carcinoma has been suggested. Of a total of 220 reported cases of hypertrophic gastritis (including the one here recorded) 6 were associated with carcinoma. In 4 of these, roentgen examination revealed a filling defect suggestive of neoplasm. Free hydrochloric acid was present in 3.

The authors' patient had been followed, with typical gastroscopic and x-ray evidence of hypertrophic gastritis, for a period of fourteen years. At the end of that time, though films did not reveal a neoplasm, laparotomy was performed because of persistent melena. This revealed a large mass 10 cm. in diameter occupying the upper third of the stomach. The resected specimen, which comprised the area of hypertrophic gastritis, was ulcerated. At the margin of the ulcer was a firm nodule, 3 × 2 cm., which was shown to be composed of bizarre malignant glandular tissue. The patient died about one year later with extensive metastatic tumor in the abdominal cavity.

Four roentgenograms; 5 photographs and photomicrographs; 1 table. JOHN F. RIESSER, M.D.
Springfield, Ohio

Massive Leiomyosarcomas of the Stomach. Report of 5 Cases. George Crile, Jr., and L. K. Groves. Gastroenterology **24**: 560–568, August 1953.

Gastric leiomyosarcomas may give bizarre manifestations. They arise from the muscular coat of the stomach and may be endogastric, intramural, or exogastric. They will often grow to large size and at the same time be difficult to localize to the stomach, roentgenologically. Lymph-node metastasis is uncommon, but the tumor will often spread to the liver and seed out on the omentum and general peritoneum. Since leiomyosarcoma is radioresistant, surgery offers the only definitive therapy. Removal of the tumor, even when it is large, is technically fairly simple, since it tends to remain encapsulated.

The authors present 5 case histories of patients with massive leiomyosarcomas. Roentgen findings were of interest. One patient was considered to have a normal stomach with displacement by an extrinsic tumor, while another, whose examination was done elsewhere, was also thought to have a normal stomach. One patient did not have preoperative roentgenograms. In the other 2 a malignant tumor was suggested. All 5 tumors were exogastric.

One patient had an exploratory operation fourteen years prior to being seen by the authors, at which time a diagnosis of inoperable tumor behind the stomach was made. Two and one-half years following treatment by the authors he was apparently well. The

massive size of these tumors suggests a long asymptomatic period or unusually rapid growth.

Four roentgenograms; 2 photographs.

JOHN F. RIESSER, M.D.
Springfield, Ohio

Giant Benign Gastric Ulcers. Richard H. Marshak, Harry Yarnis, and A. I. Friedman. Gastroenterology **24:** 339–356, July 1953.

The authors present 7 cases, with an age range from fifty to seventy-five years, in which clinical features and diagnostic studies suggested the presence of malignant ulceration of the stomach. In all 7 cases, large, benign gastric ulcers were proved to be present. All of the ulcers exceeded 3.5 cm. in diameter, the largest measuring 7.0 cm. Persistent abdominal pain, despite a rigid medical ulcer regime, was the chief complaint. Radiologically, the giant ulcers were visualized on the posterior gastric wall near the lesser curvature, above the angularis. None showed evidence of healing while the patient was under medical treatment. The roentgen examination suggested a benign gastric ulcer in 4 cases and carcinoma in 1, while in 2 the findings were reported as equivocal. This compared with a diagnosis of benign gastric ulcer in 2 out of 4 of the cases following gastroscopic examination. The only significant laboratory finding concerned gastric acidity, which in all cases was normal or elevated.

Even at the time of surgery, the ulcers had the appearance of malignant lesions, with large, hard, indurated masses involving the stomach, pancreas, and in some cases even the liver or mesentery. On gross examination, however, these were found to be completely walled-off perforations through the entire thickness of the gastric wall, and microscopic sections showed chronic benign gastric ulcer.

In the opinion of the authors, it is doubtful whether such giant ulcers could be satisfactorily controlled by means of medical therapy. They feel that subtotal gastric resection is the treatment of choice.

Twelve roentgenograms; 1 drawing.

CARLYNE A. NEWMAN, M.D.
Cleveland City Hospital

Pyloric Channel Ulcer. E. Clinton Texter, Jr., George J. Baylin, Julian M. Ruffin and Clarence W. Legerton, Jr. Gastroenterology **24:** 319–327, July 1953.

The authors evaluate statistically 55 cases of pyloric ulcer proved either roentgenologically or at operation. They point out the frequency of pyloric ulcer (38.9 per cent of gastric and 6.6 per cent of all peptic ulcers according to Brethwaite) and emphasize the wide variability of the symptoms, which are frequently misleading, bizarre, and seemingly completely unrelated to the disease.

Findings in the group here reported were: nausea and vomiting occurring within two hours after meals, in 78 per cent; atypical abdominal pain (colicky, unrelieved by eating, brought on by meals or constant), in 71 per cent; pain unrelated to meals, in 20 per cent; weight loss, in 51 per cent; typical ulcer pain, in 20 per cent; no pain, in 9 per cent. Roentgenography is considered the best available diagnostic procedure. An ulcer crater in the pylorus was found in 49 of the authors' cases, and retention in 31 cases. Other roentgenologic features relative to pyloric ulcer are lengthening, distortion, and malfunction of the pyloric canal.

Nine (16 per cent) of the patients were treated conservatively, with satisfactory results, and 26 (47 per cent) were operated upon, also with good results. Twenty (37 per cent) refused surgery and continued to have recurrent symptoms.

Surgery is considered the treatment of choice if medical management does not result in prompt recovery.

Four cases are briefly reported to illustrate the clinical and roentgen findings.

Ten roentgenograms; 2 tables.

SIMON SPENDIARIAN, M.D.
Cleveland City Hospital

X-Ray Irradiation and Conservative Surgery in the Treatment of Chronic Duodenal Ulcer. R. Kaye Scott, W. P. Holman, and E. S. Finckh. J. Fac. Radiologists **5:** 42–49, July 1953.

The authors describe their experience with x-ray irradiation in the treatment of duodenal ulcer. In patients treated by irradiation alone the results were disappointing. Combining irradiation with surgery produced more hopeful results, but these cases have not been followed long enough to be evaluated satisfactorily.

The irradiation technic consists essentially in administering to carefully defined anterior and posterior fields a gastric tissue dose of 1,500 to 2,000 r. Operation, an end-to-end antroduodenostomy, is performed two months prior to irradiation to allow adequate healing of the anastomosis. In 2 cases irradiation was done two months prior to surgery.

The gastric mucosa undergoes profound functional and histological changes following irradiation. The acid and pepsin secretions fall to low levels and there is marked cellular infiltration of the mucosa. The surface epithelium becomes flattened, and the cells of the secreting tubules undergo degeneration. With the doses employed, these changes would not appear to be permanent.

Ten cases were treated with radiation alone, and 5 of these showed relapse after periods ranging from eight to twenty-five months. Eighteen cases were treated with both irradiation and surgery. The longest follow-up in this group was eleven months, and in no instance had there been symptoms of recurrent ulceration.

Two roentgenograms; 4 photomicrographs; 2 charts; 1 photograph.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Radiological Guidance in the Surgical Treatment of Gastroduodenal Ulcer. Giancarlo Lischi. Omnia Therapeutica, Suppl. V, Pisa. (In Italian)

This little monograph deals with the role that radiology plays in the decision for surgical treatment in gastroduodenal ulcer. Six chapters consider, in turn, morphology of the ulcer, disturbances of motor function, association of other pathologic processes, postoperative gastrojejunal ulcer, and the operative indications derived from following the evolution of the ulcer by radiologic means. This last may be used as an example of the author's approach: 1. If both the radiologic and clinical picture return to normal, one is dealing with an ulcer surely benign, and conservative treatment is in order. 2. If the clinical picture is stationary but the radiologic picture better, the ulcer is in all probability

benign, but further examination is necessary. 3. If the radiologic picture is stationary and the clinical picture shows improvement, the ulcer is probably benign, but further observation is necessary. 4. If the radiologic picture is stationary and the clinical picture stationary or worse, the ulcer is probably malignant, and surgical treatment is indicated. 5. If the radiologic signs are progressive, whether the clinical signs disappear or persist, the ulcer is malignant, and surgery is indicated.

The bibliography is extensive, but mostly European.

Twenty-two roentgenograms.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

The Post-Gastrectomy Stomach Remnant. C. N. Pulvertaft. J. Fac. Radiologists **5**: 19–32, July 1953.

Some 8 per cent of Polya type gastrectomies for peptic ulceration are a failure because of postprandial symptoms which are as unpleasant as the original ulcer. There are three main groups of symptoms: those due to recurrent ulceration, those due to mechanical causes, and those resulting from alterations in the normal physiology of digestion.

Recurrent ulceration, whether gastric, stomal, or jejunal, is easily diagnosed radiologically. The recurrence rate depends on the type of primary ulceration—*i.e.*, gastric or duodenal—and on the extent of resection. It is highest in duodenal ulcers and in cases with less radical resections.

Symptoms due to mechanical causes are of two types. *Efferent loop obstruction*, usually due to adhesions between the first few inches of the efferent loop and the hilus of the spleen, causes a sense of fullness, nausea, and vomiting. *Afferent loop stasis*, with vomiting of food mixed with bile, is due at least in some cases to intermittent kinking of the loop at the stoma. Other cases are less easily explained.

Alterations in the normal physiology of digestion produce fullness, nausea, eructations, and the vasomotor symptoms of palpitations, perspiration, headache, drowsiness, and weakness. The term "dumping" is taken to imply a sensation of postprandial fullness with or without nausea or eructations, and associated with at least one of the vasomotor symptoms. These symptoms have been attributed mainly to blood sugar alterations, but mesenteric irritation due to jejunal distention and psychological factors have been mentioned as possible causes. Many other hypotheses have been advanced, and as yet there is no final agreement.

The author studied the appearance of the stomach remnant and small intestine with (1) the standard barium meal, (2) barium and hypertonic glucose, (3) barium mixed with selected foods, and (4) hexamethonium bromide.

With the standard barium meal, fullness occurs when the barium is in the jejunum, or when a slowly emptying stomach remnant distends. This technic is of little use in the investigation of the "dumping" syndrome, as the presence of food greatly modifies the behavior of the stomach.

With hypertonic glucose and barium, symptoms could be produced. The small-intestinal pattern differed from that with the standard meal, showing loss of detail due to rapid dilution of the mixture and marked increase in the rate of passage. The dilution is attributed to transfer of fluid through the intestinal wall. Serum protein estimations and packed-cell volumes showed an initial concentration of the blood.

The appearance of the stomach remnant with food and barium was also quite different from its appearance with the standard barium meal. The stomach will retain food and a quite marked sphincter-like action can be observed at the stoma. The meal leaves the stomach at various times in different patients, but it is possible to relate the time of entry of food into the jejunum to the onset of symptoms. There was no evidence to suggest that there was any dilution of jejunal contents. The optimum degree of resection, *i.e.*, one which gives a practical freedom from recurrent ulceration without increasing the incidence of symptoms, is debatable. A stomach remnant of approximately 2 inches along the lesser curvature, and 4 or 5 inches along the greater curvature, appears to give satisfactory results for duodenal ulcers.

Hexamethonium bromide depresses the motor activity of the stomach and small intestine and has a favorable symptomatic result in some cases. The meal and barium technic, used to check the effect, revealed delayed emptying of the stomach and diminished activity of the jejunum.

Twenty-seven roentgenograms; 2 diagrams; 1 table.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Obstruction of the Proximal Jejunum Following Gastric Resection and Antecolic Anastomosis. Report on Three Cases. John P. West. Surgery **34**: 98–100, July 1953.

Fortunately, obstruction of the proximal jejunal loop is probably a rare complication of gastric resection and gastrojejunostomy, having been recorded by Quinn and Gifford (California Med. **72**: 18, 1950) as occurring only 6 times in 500 resections. The author reports 3 cases and points out that in each the obstruction was due to rotation of the long proximal loop of jejunum behind the distal loop, with resulting angulation and torsion of the jejunum at the point of attachment to the lesser curvature of the stomach.

Characteristic features of this type of obstruction are: (1) constant pain; (2) vomitus which does not contain bile; (3) a roentgen shadow in the left upper abdomen due to pressure of fluid in the distended loop; (4) a mass which is palpable in thin patients; (5) perforation of the obstructed loop with the development of peritonitis.

Early recognition and treatment are the only ways to obviate a fatal outcome.

One drawing.

S. F. THOMAS, M.D.
Palo Alto, Calif.

Intestinal Obstruction with Congenital Absence of the Left Diaphragm. Robert T. Campbell. Brit. J. Surg. **41**: 56–60, July 1953.

Congenital absence of a hemidiaphragm, almost invariably the left, is a rare condition, usually incompatible with life. The great majority of patients suffer early from cardiac and respiratory difficulty, and are reported as dying in infancy or childhood. On rare occasion, a patient will achieve a balance between lung and abdominal contents and live in good health to old age.

The author presents the case of a 79-year-old man admitted with intestinal obstruction due to ileal adhesions. Previous chest films had been considered to show "eventration of the left diaphragm" with paradox-

ical motion seen on fluoroscopy. The present chest film revealed a high left hemidiaphragm and apparent pneumothorax. With continuation of colicky abdominal pain, it became evident that the cause of the apparent pneumothorax was the presence of greatly distended bowel loops in the left hemithorax. Laparotomy was performed, and the obstruction relieved. Careful examination revealed virtual absence of the left leaf of the diaphragm, with nothing to suggest rupture. Recovery was uneventful, and in the final relationship the stomach remained in direct contact with the left lung.

Eight roentgenograms. C. M. GREENWALD, M.D.
Cleveland Clinic

Intestinal Tuberculosis: Roentgenologic and Clinical Observations of an Atypical Case. Paul Eckey. Strahlentherapie **91:** 476–480, 1953. (In German)

While isolated intestinal tuberculosis in the ileocecal region is easily recognized, the disease is frequently confused with carcinoma when it occurs in other locations. This is illustrated by the following case report:

A 44-year-old female with vague abdominal symptoms had an increased erythrocyte sedimentation rate. A barium enema study revealed localized narrowing in the transverse colon, close to the splenic flexure, with absence of haustrations and a disturbed mucosal pattern, originally attributed to neoplasm. Eighteen months later, however, when there was no progression, but appearance of seven additional stenosing lesions in the small intestine, the original impression of carcinoma became doubtful. The patient was operated on, and the surgical specimens revealed ulcerative intestinal tuberculosis with productive lymph-node infiltration. The patient made an uneventful recovery from the operation and was cured with the aid of Tebethion and PAS and conservative roentgen therapy. No details of the irradiation are given.

Three roentgenograms. ERNEST KRAFT, M.D.
Newington, Conn.

Volvulus of Small Intestine Complicating Pregnancy. Report of a Case. Phillip H. Halperin, Bela Kent, and Sidney Rubin. J.A.M.A. **152:** 1219–1221, July 25, 1953.

The authors report a case of volvulus of the small bowel in a patient of nineteen years, five months pregnant, and discuss the difficulties of diagnosis. Not only are some of the normal signs of pregnancy similar to the signs observed in cases of obstruction, but roentgenographically differentiation must be made from the true ileus of pregnancy. A single scout film of the abdomen is not sufficient; a follow-up film should also be obtained, in twelve to twenty-four hours. In early volvulus the first roentgenogram may disclose no abnormality and thus be misleading. A second film will reveal the characteristic progress of gaseous distention. The prognosis is greatly improved if the volvulus is diagnosed early and surgical correction undertaken.

Two roentgenograms. MELVIN H. BECKER, M.D.
University of Michigan

Radiologic Aspect of the Small Intestine in Typhoid Fever. E. Chérigié, A. Laporte, and R. Verspyck. J. de radiol. et d'électrol. **34:** 522–523, 1953. (In French)

In an epidemic of typhoid fever in 1950, small-bowel studies were performed in 32 patients. The jejunum was dilated to two or three times normal caliber, the valves of Kerkring were enlarged and edematous, and indentations along the borders suggested the appearance of "stacked plates." Areas of spasm and puddling of barium gave a segmented aspect to the ileum. In the terminal ileum, small mucosal tumefactions about the size of a pea were thought to represent hypertrophy of Peyer's patches. Small mucosal ulcerations were also demonstrated in this area. The terminal ileum was usually least well opacified and the mucosal ulcerations were demonstrated in only 3 patients. The transit time was usually five or five and one-half hours, probably within the limits of normal. There seemed to be definite delay in dilated areas.

CHARLES NICE, M.D.
University of Minnesota

Volvulus of the Sigmoid Colon. Louis T. Gabriel, Darrell A. Campbell, and Merle M. Musselman. Gastroenterology **24:** 378–384, July 1953.

Volvulus of the sigmoid colon is one of the most frequent causes of large bowel obstruction, especially in older persons. Predisposing conditions are a long sigmoid loop and a narrow base of mesenteric attachment.

Symptoms and signs of volvulus of the sigmoid colon include abdominal pain, vomiting, obstipation, and extreme abdominal distention, this last being practically a constant physical finding.

Diagnosis can be established readily by the roentgen findings. The picture of an enormous gas-distended loop of bowel rising out of the pelvis, as shown on a scout film of the abdomen, is considered to be pathognomonic. A barium enema is indicated only as a confirmatory diagnostic measure.

The authors favor surgical treatment, since reduction either by sigmoidoscopic decompression or by barium enema will not furnish assurance of the viability of the bowel, and signs of strangulation in the old age group are particularly unreliable. Interval resection of the sigmoid colon is recommended as the best treatment.

In reporting the mortality in 21 cases of volvulus of the sigmoid colon seen at the Wayne County General Hospital, the authors consider two groups.

1. Seven patients had gangrene of the involved bowel, and 6 of these died, without surgery.
2. Of 14 patients with viable bowel, 4 died, chiefly as a result of postoperative complications.

Early diagnosis with prompt and proper management is urged by the authors.

Two roentgenograms; 2 photographs.

THOMAS HWANG, M.D.
Cleveland City Hospital

Adenomas of the Colon and Rectum. Diagnosis and Treatment in Relation to Cancer Prevention. Paul C. Morton. Ann Surg. **138:** 92–98, July 1953.

Seventeen per cent of all cancer deaths are said to originate in the colon and rectum. Many growths exist for a variable period in a precancerous state which is easily diagnosed and which can be successfully treated. Review of the literature supports the author's contention that adenomas of this region frequently become malignant. For this reason proctoscopic examination of every patient is advocated. The author pleads that all physicians be taught to do proper proctoscopic examination and that every hospital have a room for this purpose with adequate equipment. Where

adenoma is suspected but cannot be seen proctoscopically, or where additional lesions higher up are suspected, careful repeated barium enema and air-contrast examinations should be done by a competent roentgenologist.

At operation, when an adenoma cannot be found by careful external palpation of the bowel, the author does not hesitate to open the colon and insert a sterile proctoscope to search for the lesion. This assumes proper preparation with an intestinal antibiotic.

Complete excision of the adenoma should be the definitive aim of treatment. Biopsy is contraindicated. All small adenomas visible by proctoscope should be removed, with coagulation of their bases. Larger ones can be removed at the base of the pedicle by means of the electocoagulation snare loop. Many flat sessile lesions can be pulled through the loop by aid of a suction-tip funnel. Large adenomas, localized multiple adenomas, and many of those higher in the colon require local segmental resection and end-to-end anastomosis. Single adenomas of the colon beyond the reach of the sigmoidoscope may be removed through a colotomy incision, but if the lesion is large or fixed, segmental resection is necessary. Pathological examination of the specimen is necessary to establish whether the lesion is malignant or benign. If it is benign, the patient should be observed regularly for recurrences or additional new lesions. Radical excision of rectosigmoid and rectal carcinomas, along with their lymphatic drainage areas, by abdominoperineal resection is advocated.

The author concludes that, if the above fundamental concepts are followed, great strides in control of rectal and sigmoid cancer can be made.

Two photomicrographs.

GEORGE A. SHIPMAN, M.D.
New Orleans, La.

Meconium Peritonitis. T. L. C. Pratt. J. Fac. Radiologists **5**: 62–64, July 1953.

Meconium peritonitis is an uncommon condition which may be encountered in the newborn infant. It is due to the reaction of the peritoneum to the presence of meconium, a perforation in the wall of the intestine being an essential condition. Perforation can occur before, during, or after birth, as a result of atresia, intussusception, peritoneal bands, volvulus, or trauma. In many cases the cause is obscure.

Meconium ileus is associated with fibrosis of the pancreas or stenosis of the pancreatic duct. With lack of pancreatic secretion, the meconium is sticky and "putty-like" and, adhering to the walls of the intestine, produces obstruction.

Four radiologic possibilities are described:

(1) Intestinal obstruction occurring shortly after birth, where the perforation has become sealed. There are no radiologic diagnostic criteria.

(2) Calcium deposits in the peritoneal cavity with or without obstruction or evidence of intestinal perforation. Calcifications may form within twenty-four hours of perforation.

(3) A single large fluid level in the peritoneal cavity, occurring with a recent perforation.

(4) Multiple fluid levels within the peritoneal cavity, occurring with a patent perforation and a plastic peritonitis.

The prognosis is poor in meconium peritonitis Many infants are stillborn and others survive only a few days. Normal intestinal bacteria appear after three or four days of life, thus converting a sterile condition into an infective peritonitis.

The author presents two brief case reports. One patient was found at necropsy to have a severe degree of fibrocystic disease of the pancreas. Both had perforations of the colon.

Two roentgenograms; 2 photographs.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Acute Pneumocholecystitis. A Review and Report of Two Cases. Luther G. Bell, Robert B. Brown, and Harry F. Lenhardt. Ann. Surg. **138**: 268–274, August 1953.

In a review of the literature, the authors found 25 clinically proved cases of acute pneumocholecystitis, to which they add 2. The age of the patients ranged from thirty-two to seventy-four years, with the majority between fifty and seventy years. There were 21 cases in males and 6 in females. The authors quote Stevenson (Am. J. Roentgenol. **51**: 53, 1944. Abst. in Radiology **43**: 202, 1944) as to the probable course of events: (1) lodging of the stone in the cystic duct; (2) resulting decrease in local resistance of gallbladder tissues, allowing the avirulent *Cl. welchii* to become virulent; (3) increase in growth of gas bacillus in bile and gallbladder wall; (4) production of gas in gallbladder, emphysematous blebs in loose areolar connective tissue of gallbladder wall, and extension of the infection to the pericholecystic tissues.

In 8 of the cases from the literature there was associated diabetes of various degrees of severity and duration.

Clinical findings in acute pneumocholecystitis are similar in most respects to those of acute cholecystitis. Preoperative diagnosis is made by roentgenogram, which shows gas in the gallbladder or pericholecystic region. To be considered in the differential diagnosis are such conditions as perforated peptic ulcer, acute pancreatitis, pneumonia, and acute appendicitis. A case was noted in the literature of lipomatosis of the gallbladder wall which simulated pneumocholecystitis on the roentgenogram. The differentiation is possible because in lipomatosis the shadow does not change in configuration with change in position of the patient, whereas gas shadows may do so. Also in lipomatosis there is no air-fluid level.

Although the number of cases of acute pneumocholecystitis is too small to permit any adequate evaluation of the effects of treatment, the authors feel that chemotherapy should be combined with surgery.

One roentgenogram; 1 table summarizing the reported cases.

PAUL R. NOBLE, M.D
Pittsburgh, Penna.

Papilloma of the Gall Bladder with In Situ Carcinoma. Edward J. Tabah and Gordon McNeer. Surgery **34**: 57–71, July 1953.

The authors review the present rather confused situation in relation to papilloma of the gallbladder and conclude that the incidence is not as high as some writers would make us believe, since the cholesterol and inflammatory types, not being true neoplasms, should not be included under this heading. The true incidence of papilloma is believed to be approximately one in every hundred cholecystectomies.

Kirklin (Am. J. Roentgenol. **29**: 8, 1933) has out-

lined clearly the roentgen characteristics of papilloma of the gallbladder: (1) The defect in the cholecystograms maintains a more or less constant location even on repeated examinations and with the patient in different positions. (2) The defects are not infrequently two or more in number and one should not be misled into mistaking them for stones. (3) The size and location of the defects are not, as a rule, diagnostically helpful. Papillomata do not show a predilection for any particular portion of the gallbladder.

Like adenomatous polyps of the gastrointestinal tract, papillomas of the urinary bladder, and laryngeal polyps, papillomas of the gallbladder may be the site of carcinomatous change. Three of 4 cases reported here showed foci of non-infiltrating carcinoma *in situ*.

Although the authors believe that their 3 cases are the first to be recorded of *in situ* carcinoma arising from otherwise benign papilloma of the gallbladder, they strongly urge that these tumors, once they are diagnosed, be removed, because of the possibility of malignant change.

Two roentgenograms; 10 photographs and photomicrographs.

S. F. THOMAS, M.D.
Palo Alto, Calif.

Benign Non-Traumatic Stricture of the Left Intrahepatic Bile Duct. Jerome J. Weiner and Samuel LaCorte. Am. J. Surg. **86:** 120–122, July 1953.

Benign strictures of the intrahepatic ducts have not received the attention accorded to lesions of the extrahepatic ducts. Fifteen cases were reported by Carter and Gillette (J.A.M.A. **145:** 375, 1951. Abst. in Radiology **58:** 145, 1952), who are commended by the present authors for stressing the importance of cholangiographic studies in arriving at a diagnosis of these lesions, formerly relegated to the category of cirrhosis. Once diagnosis is established, appropriate surgical measures can be performed. Partial hepatectomy, with anastomosis of the dilated duct to the duodenum or jejunum, or simple dilatation of the duct may be performed.

The authors describe a case considered to be typical of gallbladder disease with progressive jaundice. The gallbladder was removed. A few days later a cholangiogram via the T-tube revealed a stenotic left hepatic duct, with the proximal portion dilated. The patient recovered uneventfully and resumed his usual routine.

The authors suggest that jaundice was the result of impaction of bile sand in a congenital stricture of the left duct, with subsequent relief due to spontaneous extrusion of the sand. They predict recurrence and state that, had cholangiography been done at the time of operation, a proper diagnosis could have been made and followed by operative correction.

Three roentgenograms.

GEORGE A. SHIPMAN, M.D.
New Orleans, La.

Percutaneous Transhepatic Cholangiography in the Diagnosis of Obstructive Jaundice. A. W. Nurick, David H. Patey and C. G. Whiteside. Brit. J. Surg. **41:** 27–30, July 1953.

The authors describe a technic of percutaneous transhepatic cholangiography. A needle 1 mm. in external diameter is inserted through the anterior abdominal wall close to the costal margin. While suction is applied, a bile duct is probed for, the needle being irrigated and redirected if, as frequently happens, a portal radicle is entered. When a bile duct is located, a few cubic centimeters of 35 per cent Diodone is injected as a check, prior to full injection of 20 c.c.

This procedure was technically successful in all 5 cases of obstructive jaundice in which it was attempted. It was unsuccessful in 3 cases without obstructive jaundice, suggesting that, without the dilatation of intrahepatic ducts from chronic obstruction, filling and roentgen demonstration of the biliary system are unlikely. In one of the 5 cases with obstructive jaundice, intraperitoneal leakage of blood and bile occurred, with death of the patient. This fatality is attributed to omission of preparation with vitamin K, which is now routine.

The test is considered comparable to other procedures, such as air encephalography, in which real dangers exist but are countered by watchful expectancy and emergency surgery if indicated. The authors feel that, in spite of the one fatality, the method is worthy of further cautious exploration as a companion test to liver biopsy in the diagnosis of obstructive jaundice.

Six roentgenograms.

C. M. GREENWALD, M.D.
Cleveland Clinic

Chemical, Mineralogic, and X-Ray Diffraction Investigation of Gallstones of Man and Cattle. W. Epprecht, H. Rosenmund, and H. R. Schinz. Fortschr. a. d. Geb. d. Röntgenstrahlen **79:** 1–20, July 1953. (In German)

Sixty cases of gallstones in man were exhaustively studied. The stones were broken up and separations were made (*a*) of the ether-soluble portion, by evaporation and recrystallization, and (*b*) the ether-insoluble portion. X-ray diffraction patterns were determined by heating in air to 500° and 1,000° C. by the Debye-Scherrer method. A smaller number of bovine gallstones were studied.

From the standpoint of structural and chemical composition the following classification is presented.

A. Homogeneous Stones
   1. Pure or homogeneous cholesterin (with or without an inorganic component).
   2. Pure or homogeneous bilirubinate (with or without an inorganic component).
   3. Pure calcium carbonate stones (rare).

B. Heterogeneous Stones
   1. Mixed stones (without or with inorganic components), in which cholesterin and bilirubinate are present in all layers and differentiation between central and peripheral layers cannot be made.
   2. Combination stones (with or without inorganic components), in which the central and peripheral layers are distinct.
      (*a*) Center, cholesterin; periphery, bilirubinate. Both center and periphery homogeneous.
      (*b*) Center, cholesterin; periphery, cholesterin and bilirubinate. Center homogeneous, periphery heterogeneous.
      (*c*) Center, bilirubinate; periphery, cholesterin. Center homogeneous, periphery homogeneous.
      (*d*) Center, bilirubinate; periphery, cholesterin and bilirubinate. Center homogeneous, periphery heterogeneous.

(*e*) Center, bilirubinate and cholesterin; periphery, cholesterin. Center heterogeneous, periphery homogeneous.

(*f*) Center, bilirubinate and cholesterin, periphery, bilirubinate. Center heterogeneous, periphery homogeneous.

Pure cholesterin and bilirubinate stones are believed to represent a metabolic disturbance. Inflammatory conditions are regarded as contributing to the deposit of inorganic calcium salts. Mixed stones are considered the result of defects in both calcium and bilirubinate metabolism, alternating or simultaneous in their effect. Many authors hold infection to be the greatest contributing cause. The writers consider it likely that gallbladder catarrh, "diathesis," stasis, and toxicity undoubtedly play a part, but the understanding of the action of these various factors is still highly debatable.

Pure cholesterin stones are practically transparent to x-rays, showing less absorption than water and soft tissues. They are therefore not seen on plain films except in rare instances in which an extremely large calculus may be questionably visualized. Stones containing significant quantities of inorganic salts, especially calcium, are visible on plain-film studies. The rather rare calcium carbonate stone is clearly demonstrated. True bilirubinate calculi are also plainly identified. Mixed and combination stones give an appearance of concentric layers, depending on the density of the materials involved. Radiating fissures are commonly seen in these stones.

The bilirubinate combinations are classified as follows:

Type 1: Bilirubinate mixed with copper and calcium combinations.

Type 2: Bilirubinate mixed with considerable calcium but very little copper.

Type 3: Calcium-bilirubinate in a crystalline form, found in gallstones from cattle but not from man.

The authors believe that their new classification, based on the constituents and their distribution within the stones, will help to clarify the roentgen findings.

Six photographs; 7 tables.

E. W. SPACKMAN, M.D.
Fort Worth, Texas

THE MUSCULOSKELETAL SYSTEM

Evaluation of the Skeletal Age Method of Estimating Children's Development. I. Systematic Errors in the Assessment of Roentgenograms. Donald Mainland. Pediatrics **12**: 114–129, August 1953.

In view of the common use of the Todd and Greulich-Pyle atlases of hand roentgenograms for estimation of bone age in children, the author presents the results of investigation of the observational error in the use of these guides. A self-trained observer made random independent unbiased assessments (knowing only the sex of each subject) of reproductions of children's hand roentgenograms as published in a current pediatric text (Macy, I. G.: Nutrition and Chemical Growth in Childhood, Springfield, Ill., Charles C Thomas, vol. 2, 1946). The Todd and Greulich-Pyle atlases were utilized as guides.

This observer tended to estimate skeletal ages lower than the values given by Macy. The error varied significantly, both between children and between roentgenograms of the same child. Comparison of expert assessments with each other showed wide variation, indicating lack of suitable stability in this type of estimation. Detailed analysis of the results is given, together with methods of statistical analysis.

The author appeals to interested readers to conduct similar surveys as outlined in the original article and to send him the results for inclusion in further study of this data.

Three tables. J. MAURICE SWAIN, M.D.
Cleveland Clinic

The Periosteopathy of Mother-of-Pearl Workers. Antonio Runco and Roberto Bossi. Radiol. med. (Milan) **39**: 643–654, July 1953. (In Italian)

Inhalation of the dust of mother-of-pearl can cause both a characteristic periosteal ossification and an allergic asthma. There are few references to this occupational disease in the literature, especially so far as the radiologic manifestations in the bones are concerned. The author describes 9 cases.

Mother-of-pearl, the shiny lining of certain mollusks, is used extensively in button manufacture. The dry shell contains between 90 and 95 per cent calcium carbonate, between 2 and 3 per cent organic substances, and between 2 and 3 per cent various mineral salts. The organic fraction belongs to the scleroproteins, being very resistant to proteolytic ferments and the action of acids and bases. It does not appear to be denatured in the body, and is probably handled as a foreign body. The authors do not exclude the possibility that there are other substances in the dust that may be harmful.

The pathogenesis of the periosteal involvement is not known. Possibly, inflammation set up in the lung has a toxic or allergic effect on the periosteum. Inflammatory foci in the lungs have been found postmortem. The process may then be analogous to pulmonary osteoarthropathy of Marie. Almost all the reported cases are in patients under twenty, though many of the workers in this industry are older. The younger the person, the more likely he is to be afflicted with a given exposure. One of the 9 patients was a child who lived near the factory. The symptoms appear after one to three years of exposure.

Periosteal biopsy shows primitive spongy bone in the early stages and condensation with stratification of the new bone in the later stages. These pictures are the same as those in Marie's pulmonary osteoarthropathy.

The first radiologic sign is a fine periosteal linear density, involving more or less of the diaphysis, with a clear zone between the area of density and the cortex, the latter remaining intact. This abnormal bony tissue then becomes much thicker, with a fluffy appearance. Within several weeks to three months from the beginning of the process, the periosteal reaction reaches its maximum. It then begins to diminish in caliber, the new-bone assuming a reticular or lamellar pattern. Finally, the periosteal new bone fuses with the cortex. This takes from months to years. There may be a slight thickening of the cortex permanently. This process is confined to the diaphysis. Usually the entire diaphysis of the short tubular bones is involved, while only a segment of the diaphysis of the long bones may be affected. In the flat bones the tendency is to involvement of the angles. The bones most commonly involved are the metatarsals (except the first), tibia, ulna, femur, radius, scapula, and mandible. The

metatarsals tend to be affected successively, beginning with the fifth. The tendency for symmetrical distribution of the lesions is only minimal.

This radiologic picture is preceded by one to four weeks by pain and tenderness to pressure in the involved part, at first moderate in intensity, but later so severe as to interfere with the patient's appetite and even sleep. A hard tumefaction appears, and the skin is red and hot. Fever is not prominent. The laboratory findings are usually normal, but at times there is a slight monocytosis. The pain disappears in one to three months. When the swelling of the soft parts recedes, the bony enlargement is palpable.

To be differentiated are trauma, calcifying hematomas, periosteal reactions secondary to soft part conditions (septic processes, angiomas, neoplasms, etc.), pulmonary hypertrophic osteoarthropathy of Marie, osteomyelitis, tuberculosis, syphilis, leukemia, bone tumors, and in the case of babies, rickets, scurvy, hypervitaminosis A, and Caffey's syndrome.

The authors emphasize that an intact cortical bone beneath the periosteal new bone is essential for the diagnosis. The prognosis is good, with or without treatment. Roentgen therapy may be of some value. The patient should, of course, be removed from the dusty environment at once.

Thirty roentgenograms; 2 photomicrographs.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

Looser Zones (Umbauzonen) of Multiple Localization and Atypical Course. Neopolo Macarini. Radiol. med. (Milan) **39**: 635–642, July 1953. (In Italian)

The author recognizes two forms of the Looser-Milkman syndrome (stripes of radiolucency in the various bones of the skeleton, often symmetrical). In one the stripes are secondary to a well defined disease, such as osteogenesis imperfecta, syphilis, hyperthyroidism, rickets, osteomalacia, sprue, chronic enteritis, or intoxication with chemicals, being due to disturbances in calcium-phosphorus-vitamin metabolism. In the second form, the stripes are also due to disturbances in the calcium-phosphorus-vitamin metabolism, but the disease entities are not well defined, and the zones of radiolucency seem to be the main abnormality. This group is made up of the hunger osteopathies and the cases described by Milkman and others. [This grouping certainly seems peculiar to this reviewer.]

The case of a 51-year-old female is reported. The past history was essentially negative, especially as to eating habits. The illness started with a severe pain in the left knee as the patient was kneeling on the floor, followed by an immediate genu valgum deformity. The physical examination revealed poor nutrition, pallor of the mucous membranes, and a left genu valgum. The basal metabolism rate was plus 17, the red cell count was 2,800,000, serum phosphatase 6.4 Bodansky units. [The calcium and phosphorus values, strangely enough, are not reported.—C.V.C.] Radiological survey of the skeleton showed radiolucent fissures in both scapulae, the right ulna, pelvis, and proximal portions of the left tibia and fibula, with medial angulation. The bones in general showed a mild osteoporosis (*sic!*), and the pelvis was deformed. After five months of treatment with calcium, phosphorus, and vitamins A and D, the fissures either disappeared or regressed, with periosteal new-bone thickening. The general condition of the patient was much improved.

This case is put into the author's second group.

Ten roentgenograms.

CHRISTIAN V. CIMMINO, M.D.
Fredericksburg, Va.

Osteochondritis Deformans Coxae Juvenilis. Familial Demonstration. W. R. Hamsa and L. S. Campbell. Am. J. Dis. Child. **86**: 54–59, July 1953.

The case histories of three brothers with coxa plana are given. In 2, symptoms developed at the age of eight, in the third at eleven. The involvement was bilateral in the first child, unilateral in the others. Because of the familial incidence, the authors suggest that inherent hip weakness or a familial endocrine change may be a predisposing factor.

Six roentgenograms. ARTHUR S. TUCKER, M.D.
Cleveland Clinic

Histiocytosis X. Integration of Eosinophilic Granuloma of Bone, "Letterer-Siwe Disease," and "Schüller-Christian Disease" as Related Manifestations of a Single Nosologic Entity. Louis Lichtenstein. Arch. Path. **56**: 84–102, July 1953.

The author critically reviews the literature which has accumulated since his previous report in 1944 (Jaffe and Lichtenstein: Arch. Path. **37**: 99, 1944) and amplifies the concept that the common pathologic denominator of eosinophilic granuloma, Letterer-Siwe disease, and Schüller-Christian syndrome is a distinct and apparently specific inflammatory histiocytosis, whatever its etiologic agent may ultimately prove to be.

In actual practice it is not always possible on the initial examination of the patient, especially a young child, to be certain that skeletal lesions identified as eosinophilic granuloma do not have some extraskeletal histiocytic counterpart not yet manifested. Thus it follows that from the roentgenogram of such a lesion, or even from a biopsy of it alone, one cannot make a reliable prognosis, since this depends largely upon the presence or absence of associated visceral involvement and more particularly upon the distribution, extent and severity of possible visceral lesions. The author cites a case in which, many months after the appearance of multiple areas of bony involvement diagnosed on biopsy as eosinophilic granuloma, it became evident that the patient was suffering from Schüller-Christian disease. Terminally constitutional and visceral changes developed, with production of a clinical picture of so-called Letterer-Siwe disease (acute or subacute disseminated histiocytosis X).

As a result of the analysis of the literature and a review of his own cases, the author finds it increasingly evident that the initial pathologic picture is essentially that of an inflammatory histiocytosis (or granulomatosis), which may or may not be accompanied by intense eosinophilic reaction, but which, in any event, exhibits no significant tendency at the outset toward lipidization. The picture of lipogranuloma, so-called, apparently represents the late or end phase of the evolution of the histiocytic lesion. It is felt that the undue prominence given this phase of this disease complex in the past is due to the observations having been made largely at autopsy and that with the increasing use of biopsy material the varying tempo of the disease complex can be better appreciated.

One roentgenogram; 6 photomicrographs; 2 tables.

RICHARD E. OTTOMAN, M.D.
Los Angeles, Calif.

Neurilemmoma of Bone. H. Morus Jones. Brit. J. Surg. **41**: 63–65, July 1953.

A case of neurilemmoma primary in a metacarpal bone is reported. The lesion probably arises from the sheaths of the sparse nerve trunks within the marrow cavity which accompany the nutrient vessels into bone. Such an origin tends to account for localization of the tumor to the midshaft region of long bones, where cystic expansive rarefaction is seen. The clinical course appears to be benign; local removal is adequate, and prognosis good.

The author's case is apparently the sixth to be reported in bone. The patient was a 44-year-old woman referred to the hospital with a swelling on the back of the left hand which had been considered a "ganglion." It was fluctuant and could be transilluminated. Clinically and radiologically the tumor was thought to be a chondroma. It was removed, and a bone graft was inserted. Pathologic examination and comparison with the previously reported examples led to the diagnosis. Although osseous changes occur in the neurofibromatosis of von Recklinghausen, in none of the cases of neurilemmoma were stigmata of that disease present.

Four roentgenograms; 1 photomicrograph; 1 drawing, in color; 1 table. C. M. GREENWALD, M.D.
Cleveland Clinic

Skeletal Changes in Parathyroid Tetany. W. Achenbach and A. Böhm. Fortschr. a. d. Geb. d. Röntgenstrahlen **79**: 95–103, July 1953. (In German)

Tetany in hypoparathyroid conditions, although not fully understood, is considered to be due to a disturbance in balance of electrolytes (Ca-, P-, K-, and H-ions). In so-called "idiopathic" tetany, an absolute or relative parathyroid insufficiency is assumed. Bone constitutes the great reservoir of calcium; as the content is increased in the bony structures, there is a decrease in the blood with a relative increase of phosphorus.

On the basis of a case which they report in full and a review of the literature, the authors enumerate the skeletal findings. In the skull there are thickening of the inner table (hyperostosis frontalis) and homogeneous thickening of the petrous portion of the temporal bone. The facial bones may be thickened to lesser degree. The spine in general shows normal calcification, but there may be a heavy double line of sclerotic bone about the borders of the vertebral bodies. The hip joints are deformed, with thickening and sclerosis of the femoral head, neck, and acetabulum and narrowing of the joint space. There is frequently calcification of the joint capsules. Sclerosis of the iliac borders is observed, with an underlying zone of rarefaction. Similar changes are present about the shoulders and knees, with increased density and thickening of the cortex, narrowing of the marrow spaces and occasionally of the joint spaces. The hands are short and plump, showing osteophytic outgrowths, broadening of the phalangeal and metacarpal bases, and coarsening of the bony trabeculae with vacuolization.

The more severe cases, (hypoparathyroid cretinism) show retardation of growth, calcium deposits in the brain stem and choroid plexus, poor dentition with early loss of teeth, severe trophic disturbances of skin, hair, nails, etc., and deficient mental development.

Eight roentgenograms. E. W. SPACKMAN, M.D.
Forth Worth, Texas

A Bone Disorder Associated with Copper Deficiency. I. Gross Morphological, Roentgenological, and Chemical Observations. James H. Baxter and Judson J. Van Wyk. Bull. Johns Hopkins Hosp. **93**: 1–13, July 1953.

After periods of two to four months, more than two-thirds of a group of dogs on a copper-deficient diet were observed to be lame or to have deformities of the extremities. The forelegs were bowed outward at the elbows, while the hind legs were bowed inward with the knees held together for support. The ends of the bones at the wrists and elbows often became large and "knotty." Hyperextension of the wrists was a prominent feature, leading to a decrease in height of the anterior end of the animal and giving it the appearance of walking on snowshoes. Obvious fractures of the limbs, sometimes multiple, occurred.

A study of roentgenograms and of the bones themselves showed a characteristic decrease in thickness of the cortices. This change was observed most consistently in the shaft of the femur, but it was prominent also in the metacarpals and other limb bones. The decrease in thickness of cortical bone in the deficient dogs was associated with an apparent increase in volume of the marrow. The marrow was pale and contained less fat than normal. Trabeculae often were lacking throughout the diaphyses, and the metaphyses were of diminished extent. In addition, the trabeculae of the metaphyses of the limb bones, of the epiphyseal centers of ossification, and of such bones as the ribs and vertebrae were more delicate, of smaller caliber, and more widely spaced than normal. This deficiency of trabeculae was partially responsible for the "ground-glass" appearance of the bones in roentgenograms. This latter alteration was most readily visualized in the centers of ossification of the distal radial epiphyses and other small bones about the wrist and in the femurs. The bones of the copper-deficient animals were as mature as those of control animals, judging by the maturation of centers of ossification of the epiphyses. There were also a number of changes which were interpreted as effects of stresses acting on abnormally weak bones; these included bowing, compensatory thickening, fractures, displacement and distortion of epiphyses, and longitudinal compression with lateral expansion of the ends of limb bones. This last change probably accounted for the enlargement and hyperextension of joints.

Anemia and graying of the hair accompanied the bone changes. The disorder was relieved by the administration of copper, and never occurred in control animals. Calcium, inorganic phosphorus, and vitamin D levels of the serum of copper-deficient animals were normal, and the bone lesion was not characteristic of rickets. The bone disorder was not a result of inactivity, inanition, or anemia. The failure of iron deficiency, with severe anemia, to produce similar changes indicated that the bone disorder of the present study was a specific effect of copper deficiency.

Seven roentgenograms; 7 photographs; 1 chart; 3 tables.

Lumbosacral Junction: Roentgenographic Comparison of Patients With and Without Backaches. Clarence A. Splithoff. J.A.M.A. **152**: 1610–1613, Aug. 22, 1953.

Roentgen findings in 100 consecutive patients with lumbar backache seen at the New York Orthopedic Dispensary and Hospital, exclusive of those with ob-

vious herniated disks, tuberculosis, or other disease, and 100 adults without backache were compared. Anteroposterior, lateral (with thighs extended), and 45-degree cephalad-oblique views were utilized to visualize the fifth lumbar intervertebral disk and facets.

No significant correlation was found between backache and spondylolisthesis, congenital deformity, posterior displacement of the fifth lumbar vertebra, width of the fifth lumbar disk, or position of the lumbosacral facets.

The angle of the sacrum was slightly less in the group with backache, indicating either muscle spasm or a voluntary flattening of the back. Osteoarthritis was also slightly more frequent in this group. The commonest position of lumbosacral facets in both groups was in the coronal or anteroposterior plane.

The fact that the findings in the two groups so closely parallel each other does not discount the importance of routine roentgenographic study in all cases of chronic backache.

One line drawing; 3 graphs; 3 tables.

FRED J. HODGES III, M.D.
University of Michigan

Osteitis Pubis of Traumatic Etiology. Raymond J. Adams and Fremont A. Chandler. J. Bone & Joint Surg. **35-A:** 685–696, July 1953.

Osteitis pubis is generally considered to be an inflammatory disease of bacterial origin. It occurs most commonly following retropubic surgery in males. In cases studied by biopsy, the histologic findings have been those of inflammation. In all reported cases in which a culture has been taken, bacterial growth has been obtained.

The authors report a case, apparently of traumatic origin, in an eighteen-year-old male. The patient complained of lower mid-abdominal pain which had been present for a year, having started shortly after he had run on an indoor track. Roentgenograms showed a destructive lesion in the region of the symphysis pubis. Histologic study of biopsy material showed degeneration of hyaline cartilage, fibrosis of marrow spaces, and a low-grade inflammatory reaction in the connective tissue and synovial membrane. The inflammatory reaction was characterized by lymphoid cell, plasma cell, and mononuclear cell infiltration. These findings the authors regard as "suggestive evidence that the traumatic disease involving this patient's symphysis was inflammatory in nature and very possibly bacterial in origin."

There were found in the literature reports of 4 cases of osteitis pubis in young males following trauma. The roentgenographic changes are those of decalcification of the bodies of the pubic bones, progressing to produce definite bone defects and, at times, sequestra.

A study of the pubic bone is helpful in explanation of the occurrence of osteitis in these younger patients. While the external aspect and the pubo-ischial rami are well covered by muscle attachments, a large area of the internal aspect is covered only by periosteum and parietal pelvic fascia. Because of this, the blood supply to the cortex is less than at bone surfaces with muscle attachments. At around twenty years of age there is considerable physiologic bone absorption involving the bony surfaces at the symphysis. It may be postulated that trauma, either directly to the symphysis or through tension of the rectus abdominis muscles, causes periosteal and subperiosteal damage to bone. If this occurs at a time when bone structure is weakened by physiologic bone absorption, the effect may be more damaging than otherwise. Thrombo-embolic occlusion of the end vessels of the symphysis may then occur and a subsequent transient bacteremia may establish a focus of infection.

The authors feel that "traumatic osteitis pubis" represents a definite disease entity.

Seven radiographs; 5 photomicrographs; 1 table.

RICHARD P. STORRS, M.D.
Syracuse, N. Y.

A Survey of Carpal and Tarsal Anomalies. Ronan O'Rahilly. J. Bone & Joint Surg. **35-A:** 626–642, July 1953.

This is a comprehensive classification of anomalies of the human carpus and tarsus. These anomalies are of frequent occurrence and are of considerable interest in their teratologic, embryologic, roentgenologic, orthopedic, and medicolegal aspects. There are five chief categories: (1) severe anomalies associated with antebrachial (crural) or digital anomalies, or both; (2) fusions; (3) accessory ossicles, including sesamoids; (4) bipartition; (5) structural alterations associated with such deformities as talipes.

The first four categories are discussed in some detail. A bibliography of 127 references is given.

Seven figures (diagrammatic representations of anomalies).

RICHARD P. STORRS, M.D.
Syracuse, N. Y.

Fracture of the Carpal Navicular (Scaphoid) Bone: An End-Result Study in Military Personnel. Joseph S. Barr, William A. Elliston, Henry Musnick, Thomas L. Delorme, Joseph Hanelin, and Arthur A. Thibodeau. J. Bone & Joint Surg. **35-A:** 609–625, July 1953.

This study was undertaken to determine the incidence of fracture of the carpal scaphoid in the Armed Forces, the amount of disability arising from both united and ununited fractures, the factors predisposing to non-union, and the results of various methods of treatment of non-union.

The group studied consisted of 44 veterans with united or ununited fractures incurred at least five year earlier. The small number of patients made statistical observations valueless, but a clinical survey was undertaken. Each patient was studied by a team consisting of an orthopedic surgeon, a roentgenologist, a psychiatrist, and a physician interested in muscle physiology. A history was taken and compared with health and hospital records. Further study included physical examination, psychiatric evaluation, ergographic studies for determining the work capacity of the hand, and detailed roentgenographic examinations.

The following observations were made:

1. When healing by primary union occurs in fractures of the carpal scaphoid, there is minimal residual disability and no interference with the performance of duty.

2. Approximately one-half of patients with ununited carpal scaphoid fractures were surveyed out of the service because of wrist symptoms. The presence of non-union established before induction should disqualify an individual for full-duty status.

3. Non-union occurred in 22 per cent of the cases studied. The factors apparently tending to result in non-union were insufficiently prolonged immobilization and delay in instituting treatment.

4. The results of treatment of non-union in this series of cases were not satisfactory. Of 14 cases, 9 were treated conservatively without improvement. Five were treated operatively, with a satisfactory result in 2.

5. Those cases with non-union showed partial absorption, displacement, and fragmentation of the fragments, cyst formation in the navicular, and degenerative arthritic changes. All cases showed impaired wrist function, but earning power was impaired in only 42 per cent.

6. Study by a specially designed recording ergograph showed little or no impairment of work capacity in cases with primary union. Of 14 patients with non-union, only 1 showed normal work capacity in the wrist involved.

Seventy roentgenograms; 3 photographs.

RICHARD P. STORRS, M.D.
Syracuse, N. Y.

GYNECOLOGY AND OBSTETRICS

A New Instrument for Hysterosalpingography. Lennart Kjellman. Acta radiol. **40:** 35–38, July 1953.

The author describes a new device for the introduction of the contrast medium in hysterosalpingography, based on the principle of suction attachment of the instrument by means of a basin-shaped bell of plastic. A cone of plastic, measuring 1.0 cm. in length and of suitable diameter for the particular case, is screwed into the center of the basin. The plastic basin is tightly fitted to two stainless steel tubes, which insert into its sides. One tube is attached to a vacuum pump and the plastic is drilled so that this connects to the hollow basin portion causing it to remain tightly adherent to the cervix. The opposite tube connects with the central cone, and through this the contrast material is injected.

Two salpingograms; 1 photograph.

GEORGE REGNIER, M.D.
University of Arkansas

The Advantages of X-Ray Diagnosis in Pregnancy. Robert H. Barter and John Parks. South M. J. **46:** 555–561, June 1953.

The purpose of this paper is to indicate instances in which the use of diagnostic roentgenology can be of value to the clinician in his care for the maternity patient and the unborn fetus.

Though ideally roentgen pelvimetry should be done in all primigravid patients at term, practically it may be limited to those having abnormal clinical findings upon manual mensuration, namely, (*a*) a diagonal conjugate of 11.5 cm. or less, (*b*) a reduction in the interspinous and intertuberous diameters, (*c*) abnormal curvature or forward displacement of the sacrum, and (*d*) a masculine type pelvis. The procedure is indicated, also, in all primigravid patients with a breach or other abnormal presentation; in any primigravida with an unengaged fetal head at term; in all patients five feet or less in height; in all patients with abnormal presentations, particularly if the fetus is estimated to be larger than any previous baby the patient has had; in any patient with a history of difficult labor; in any patient who is to be allowed a trial labor after having had previous uterine surgery; in any patient who is progressing poorly in labor; in any patient with a history of fracture of the bony pelvis; in any patient classified as having true uterine inertia (cephalo-pelvic disproportion to be excluded).

The exact method of x-ray mensuration is not considered of extreme importance, since most methods have been shown to be reasonably accurate. The authors believe that a method should be used which gives a true view of the pelvic inlet (as that of Isaacs: Am. J. Roentgenol. **63:** 669, 1950. Abst. in Radiology **56:** 623, 1951), since the morphology of the inlet usually indicates the obstetrical characteristics of the entire pelvis. Actually a mid-pelvic capacity is of greater importance than the inlet measurements, inasmuch as this capacity is almost always less than that of the inlet (except for the rachitic pelvis, where the capacity may be actually greater). Pelvic capacity is satisfactorily measured by the Mengert method (J.A.M.A. **138:** 169, 1948. Abst. in Radiology **53:** 298, 1949).

Among other applications of roentgenography in obstetrics, are establishment of pregnancy, especially after the sixteenth to eighteenth week of gestation, when the skeletal parts become visible; establishment of cephalopelvic proportions, as well as the presentation and position of the fetus; antepartum diagnosis of multiple pregnancy; differentiation, in the presence of an overdistended uterus, between polyhydramnios, a single large fetus, and multiple gestation; estimation of fetal age after the thirty-fifth week of gestation, by the visibility (in 95 per cent) of the epiphysis of the distal end of the femur; antepartum diagnosis of fetal abnormalities (anencephaly, microcephaly, hydrocephalus), especially if the uterus is too small or too large for its gestational date; fetal death *in utero;* antepartum diagnosis of fetal hydrops, most commonly due to erythroblastosis; diagnosis of abdominal pregnancy. Incidental findings include calcified myomata, ovarian tumors, maternal anomalies, calculi and physiological abnormalities of the urinary tract. Placentography has its greatest usefulness in the presence of bleeding in the last trimester.

If the best results are to be achieved from the use of diagnostic roentgenology in pregnancy, the roentgenologist and obstetrician should work as a team, combining the roentgenologic data with the clinical findings.

Nine roentgenograms.

RICHARD E. OTTOMAN, M.D.
Los Angeles, Calif.

Habitual Abortion: A Radiographic Technique to Demonstrate the Incompetent Internal Os of the Cervix. Frank E. Rubovits, Norman R. Cooperman, and A. F. Lash. Am. J. Obst. & Gynec. **66:** 269–280, August 1953.

A number of representative cases of incompetence of the area of the internal cervical os is presented. Such patients are usually found to have had two or more second-trimester abortions. The majority have a history of some trauma to the uterine cervix. They reveal no significant general physical abnormality but show characteristic radiographic changes in the area of the internal os.

Radiographic studies are important in establishing the presence of an incompetent internal os and thus permitting surgical correction. In 3 of the 5 cases reported here, the patients were able to progress to term or near-term deliveries, following corrective measures.

The authors' technic includes first the introduction of a Best-Mixter cannula so modified that the outer caliber is the same throughout. A small balloon is attached to

the tip of the cannula and a fluid radiopaque medium, such as Urokon, is introduced. Serial films are taken with the medium in the balloon and at intervals during its removal. Following withdrawal of the balloon, a Rubin cannula is inserted into the cervical canal, 4 to 5 c.c. of Lipiodol are instilled, and films are obtained simultaneously with withdrawal of the cannula and after its removal.

Twenty-six roentgenograms; 6 drawings.

ROBERT L. EGAN, M.D.
Jefferson Medical College

Unruptured Tubal Term Pregnancy. Kurt G. Fraehtman. Am. J. Surg. **86:** 161–168, August 1953.

The usual outcome of tubal pregnancy is rupture of the tube, but rarely the fetus is carried to term. In the 74 cases of unruptured tubal pregnancy previously reported and tabulated here, the fetal mortality was 80 per cent. The last known maternal fatality occurred in 1937. Up to that time, the mortality among 43 women with dead fetuses was 28 per cent, and among 13 with live fetuses 38 per cent. Seventeen patients have been successfully operated on since 1938, and in 3 cases living infants have been obtained.

The author reports a case in which fetal movements stopped at about term and spontaneous onset of labor was awaited for six weeks. At that time surgery was decided upon. On clinical examination, it was thought that the uterus could be palpated under the fetal head, but hysterography and cystography seemed to indicate an intra-uterine pregnancy. At surgery the fetus and placenta were found to be completely enclosed in the left tube, with the uterus enlarged to about the size of a three months gestation. The fetus was dead but fully developed, weighing four and one-half pounds.

Inspection of the operative specimen explained the seemingly incorrect roentgen findings. The uterine fundus being displaced to the right side, the wide opening of the dilated left tube was in direct continuity with the cervical axis. The contrast medium reached up toward the opening of the tube and almost touched the fetal head, giving the erroneous impression of an intrauterine pregnancy, and no further attempt was made to visualize the entire uterine cavity. The author warns that "uterograms done with small amounts of contrast medium may give the erroneous impression of an intrauterine pregnancy, due to a wide direct communication of the dilated tube with the uterine cavity."

Two roentgenograms; 3 photographs; 1 photomicrograph; 1 table.

ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Radiological Determination of Placental Site. S. J. Boland. Irish J. M. Sc., July 1953, pp. 274–278.

The thickest part of the uterine wall as seen roentgenologically is a composite of uterine muscle, placenta, and liquor amnii. To demonstrate the placental soft-tissue shadow, the author obtains three roentgenograms: an anteroposterior supine, a lateral supine, and, when the patient's condition permits, the opposite lateral in the erect position. Roentgenograms of high quality are, of course, essential. The author uses a Siemens 4-valve unit and air-cooled rotary anode tube, with the following factors: 65 to 75 kv., 300 ma., distance 100 cm., 0.3 second.

Eighty-two patients with hemorrhage occurring after the thirtieth week of pregnancy were examined. In 73 cases, the position of the placenta was determined, and the diagnosis was confirmed clinically at a later date. In 7 cases, the placenta was not visualized and in 2 cases placenta praevia was incorrectly diagnosed.

In this series, the placenta was visualized on the posterior wall in 23 patients and on the anterior wall in 20 patients. It could be seen on the lateral wall 9 times and in the fundus on 4 occasions. A diagnosis of placenta praevia was made 17 times.

Eight roentgenograms.

DEAN W. GEHEBER, M.D.
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THE GENITOURINARY SYSTEM

Measurement of Contrast Medium Reflux in Retrograde Pyelography by Means of Blood Pyridoniodine Determination. G. Möckel and K. Gaede. Schweiz. med. Wchnschr. **83:** 697–702, July 25, 1953. (In German)

In retrograde pyelography possibilities of harm to the patient lie in mucosal trauma, tissue trauma, introduction of infection, and reflux. Modern non-toxic contrast media endanger chiefly through infection, with the magnitude dependent upon both the quantity and the location of the reflux. Tubular, interstitial, pyelovenous, and pyelolymphatic extrusions are recognized types. Reflux produced by excessive pressure may be avoided in the normal patient with careful technic. In most instances, however, reflux is a part of the disease, with the dye entering the tubules by reason of changes in the papilla, passing into the veins in certain cases of hematuria, going into the tissue through epithelial defects in infection and trauma, or getting into tissue that has been weakened by tumor.

Using the Pyridoniodine method of Apert, the quantity of contrast medium in the blood can be determined; this supplements the roentgenogram as a chemical measure of reflux. Eighty successive patients were studied with blood iodine levels taken at two, three, five, fifteen, and thirty minutes. Included were normal subjects and those with pyelonephritis, tuberculosis, lithiasis, tumor, hydronephrosis, and idiopathic hematuria. The injection pressure was rigidly controlled.

In terms of the blood levels, the 80 cases fell into three groups: those with normal pyelograms and a low blood iodine; those with such conditions as a hydronephrosis, a stone, or a blockage of the drainage system, and a moderately elevated iodine content; and those patients with a markedly elevated iodine level. The high blood values in this latter group were associated in some instances with radiographic pyelovenous reflux. There were three exceptions, in patients with hematuria, in which no sign of reflux could be detected on the films. One must here assume a direct connection with a vein which permitted the dye to enter the circulation so quickly that it was never in sufficient concentration to cast a shadow.

All cases exhibited a minimal iodine rise indicative of straight absorption by contact with the mucosa. Pyelotubular and interstitial reflux produced negligible elevation above the base values in the completely normal patients. Pyelolymphatic reflux was associated with a moderate rise and with the highest Pyridon reading in the five minute specimen.

Four roentgenograms; 2 graphs; 1 table.

WILLIAM F. WANGNER, M.D.
Royal Oak, Mich.

The Radiologic Appearances of Diverticula of the Male Cavernous Urethra. Nils P. G. Edling. Acta radiol. **40**: 1–8, July 1953.

In discussing diverticula of the cavernous urethra, it is necessary to separate cavities due to cyst formation of the bulbo-urethral (cowperian) ducts and those caused by a congenital disturbance in the closure of the urethral channel. The former are situated in the posterior portion of the cavernous urethra, the latter in the penile urethra.

It may be difficult to determine even pathologically if a paraurethral cavity is congenital or acquired. Undoubtedly, in small children paraurethral cavities are of congenital origin, and in cases of cyst formation of the bulbo-urethral ducts due to retention of secretions, to folds or adhesions, that cause prestenotic dilatations. In adults, cyst formation may be a sequel to circumscribed postinflammatory or posttraumatic shrinkage with stenosis of the ducts and prestenotic dilatation.

The author collected 31 cases for further roentgenologic studies: 27 in the posterior portion of the urethra and 4 in the anterior portion. In 24 of the first 27 cases, the diverticula were situated in the lower part of the urethra, where the bulbourethral ducts open, or in the ducts themselves. At the completion of the examination they emptied with the urethra. The outlines in all cases were smooth and regular. The smallest cavity was 1 × 6 mm., the largest 30 × 60 mm. In 10 of these 24 cases there was contrast filling, not only of the cavities, but also of the proximal part of the bulbourethral duct, and, in some cases, of the bulbourethral gland. These 10 cases indicated that the diverticula were local dilatations of the bulbourethral ducts. There is in general no difference in the appearance of the cavities in the 14 cases in which the ducts were not completely contrast-filled. In the remaining 3 of the first 27 cases, only the bulbourethral duct on one side was contrast-filled and dilated.

In all of these 27 cases there was a history of lower urinary tract symptoms. In some cases certain transurethral procedures had been carried out. The possibility, therefore, of trauma or infection cannot be excluded as a cause of diverticulum formation. However, if an acquired lesion has been present, it has led to one or more circumscribed narrowings of the bulbourethral ducts followed by secondary prestenotic dilatations. The dilatation of the end part of the bulbourethral duct in 2 of the cases seemed to demand a special explanation. Here the cause would seem to be either a congenital malformation or a dilatation due to a disturbance in the closing mechanism of the end part of the ducts.

The remaining 4 of the 31 cases had a different character, site, and shape. The cavity was situated in the penile part of the urethra in all cases; the contours were smooth and the shape rounded, oval, or in the form of a local urethral dilatation. Despite histories of trauma and infections, the appearances suggested preformed cavities or dilatations.

The different forms of diverticula of the cavernous urethra described above may be difficult to separate from other paraurethral changes. Dilatations of the bulbourethral ducts due to spread of post-inflammatory changes in the walls may cause confusion. However, the width of the lumen and the contours are often irregular. The ducts do not end in smooth cavities but open directly into the urethra, usually by a visible communication. In addition, the urethra often has an irregular lumen due to strictures and to small paraurethral cavities.

Paraurethral cavities due to fistula formation must also be kept in mind. If irregular passages are closed, a paraurethral cavity may remain. The history and irregular walls help to avoid a wrong diagnosis.

Small urethrocavernous refluxes sometimes appear in the lower wall. The urethral border of the reflux shows a smooth outline without any visible communications.

False passages in the region must also be considered. There is generally a history of trauma, and the false passage is demonstrated as an irregular ramification or bulging from the urethral shadow, in visible communication with the urethra.

Sixteen roentgenograms.

JAMES R. MORRISON, M.D.
University of Arkansas

THE BLOOD VESSELS

Translumbar Aortography as a Diagnostic Aid in Localizing Arterial Emboli. Charles G. Lovingood and Richard Patton. Arch. Surg. **67**: 164–172, August 1953.

The authors describe the advantages of translumbar aortography for the localization of arterial emboli prior to embolectomy. The procedure requires only a few minutes and permits a positive diagnosis, including the exact site of the obstruction. When the embolus is accurately localized, the incision can be accurately placed for its precise exposure.

Five cases are described, all of which showed correct localization of the embolus. Embolectomy was done in 4 patients.

Five roentgenograms. RICHARD A. ELMER, M.D.
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Thrombosis of the Abdominal Aorta Treated by Thrombo-Endarterectomy. A Case Report. John P. West, Charles F. Schetlin, and Frederick J. Schilling. Ann. Surg. **138**: 259–261, August 1953.

In a brief history and discussion of thrombosis of the abdominal aorta, the value of aortography is emphasized. Early diagnosis can be made by this method, before there is complete occlusion of the vessel. The authors feel that the hazards of thrombo-endarterectomy are lessened in this way. Their technic is not discussed.

The case presented is that of a 37-year-old woman who experienced progressive pain and fatigue in the right calf, finally involving both legs. On physical examination, pulsation was found to be diminished in the right extremity. An aortogram revealed an irregular filling defect in the right side of the aorta at the bifurcation, extending into the right iliac artery. The patient was explored through a right paramedian incision, and a thrombus was found on the right lateral and posterior wall of the terminal aorta, occluding approximately 75 per cent of the lumen. A smaller thrombus was present in the right iliac artery. Both were removed, with the involved intima. One month later pulsations were good in both extremities, and an aortogram showed essentially normal flow through the aorta and iliac vessels. The patient was able to walk eight or more blocks without undue fatigue.

Two roentgenograms; 1 drawing.

ALLIE WOOLFOLK, M.D.
Pittsburgh, Penna.

Ascending Erect Phlebography. E. M. Colvin and J. Frank Walker. J.M.A.Georgia **42:** 343–346, July 1953.

The authors have used the ascending erect phlebography technic as described by Scott and Roach (Ann. Surg. **134:** 104, 1951. Abst. in Radiology **58:** 909, 1952) on 75 patients during the past year. This method provides satisfactory filling of the deep venous system of the leg and demonstrates the presence or absence of competent valves. The procedure tests the antigravity or functional mechanism of the valves and produces maximal distention of the valve sinuses. It affords a high intensity of radiographic contrast which not infrequently is unsatisfactory with phlebography in the horizontal position.

The patient is placed on the radiographic table, which is tilted 75 to 80 degrees. The leg is internally rotated 25 degrees to minimize superimposition of bony and vascular structures. A tourniquet placed immediately above the ankle obstructs the superficial venous flow. Following insertion of a 20-gauge needle into a superficial vein of the foot, 25 to 50 c.c. of 35 per cent Diodrast is injected rapidly but not forcibly over a five- to ten-second period. Films of the leg and thigh are then taken. Preliminary sensitivity tests are done.

The procedure is not recommended in the usual varicose veins of the extremity unless doubt as to the status of the deep venous system exists or a definite history of thrombophlebitis is obtained.

Seven roentgenograms. JOHN S. SCHLECHT, M.D.
Cleveland City Hospital

Percutaneous Splenic Venography. V. Gvozdanović, E. Hauptmann, E. Najman, and B. Oberhofer. Acta radiol. **40:** 17–26, July 1953.

Surgical procedures for relief of portal hypertension are facilitated if the operator is forewarned as to the presence and site of disturbances in circulation, particularly thrombosis of the splenic vein.

The authors describe their technic for splenic venography. The spleen is punctured percutaneously, with a lumbar puncture needle directed obliquely toward the hilus, 20 c.c. of a 70 per cent solution of the contrast agent (Joduron) being injected within three to five seconds. The first film is obtained after 10 c.c. of the medium has been introduced, the second toward the end of the injection, the third after its completion, and the fourth two or three seconds later.

The authors' series consisted of 14 cases, in 9 of which the findings were normal. In 1 case thrombosis of the splenic vein was discovered, and in this instance the actual site of the obstruction was demonstrated, as well as an extensive network of collaterals. In 2 cases there were extensive esophageal and gastric varices without disturbances of the splenic circulation. It was observed that the medium left the spleen only as long as there was an elevated pressure incident to the injection. After completion of the injection, the medium no longer enters the portal circulation in any radiologically demonstrable quantity. That remaining at the site of the injection disappeared within one hour.

No serious complications resulted in this series, though the procedure is not considered entirely free from danger. In those patients subsequently operated upon it was impossible to detect injury to the capsule or parenchyma in the punctured area.

Eleven roentgenograms.

H. WENDELL WARD, M.D.
University of Arkansas

Vasographic Contribution to the Etiology and Genesis of the Crural Ulcer. E. Vogler. Fortschr. a. d. Geb. d. Röntgenstrahlen **79:** 79–94, July 1953. (In German).

Because of its chronic course and tendency to recur, crural ulcer is a most difficult lesion to treat. Its relationship to vascular changes has long been recognized. It is considered by many observers to be the end-result of the "varicose syndrome." Recent studies have indicated that both arterial and venous changes affect the capillary bed within the involved tissues and result in nutritional disturbances. The author has originated a new method of vasography to investigate this problem.

Under local anesthesia, the femoral artery is punctured. Any of the commonly employed intravenous contrast materials may be used, but those with an iodine content of 70 per cent are diluted with normal saline to make a 50 per cent solution. For the first injection, 17 c.c. of contrast material is used and a film is obtained to demonstrate the arteries of the thigh. After about seven minutes, a second injection is made, using 40 c.c. Films are obtained of the involved area after each 5 or 10 c.c. has entered the vein. The first five to seven films are made at intervals of three seconds, the subsequent films at intervals of five to ten seconds. The course of the medium is followed through the arterial, capillary, and venous phases during a period of approximately twenty-eight to thirty-four seconds. In some cases, following the second series, 2 c.c. of Hydergin, diluted to 20 c.c. by normal saline, was injected and a further series of films was taken. No untoward effects have been observed in the author's series.

Arterial disturbances, usually in the anterior tibial, were observed in practically all of the 73 cases studied by the author. In the early stages, changes were spastic in nature, later organic, in a large proportion of cases involving the main stem artery rather than the precapillary branches exclusively. Arteriovenous anastomosis was at first suggested by premature filling of the smaller veins. This premature filling was later visualized directly, and shunting of blood from artery to vein was demonstrated without filling of the capillary bed. Arteriovenous anastomoses were present in over 50 per cent of the author's cases, both at the site of the ulcer and over a more diffuse area. Comparison of films before and after administration of Hydergin showed a normalizing effect on the circulation in the precapillary bed. There was less shunting of blood through the arteriovenous channels and better blood supply to the capillaries. Varicosities and phlebectasia were proved in 72.6 per cent of the cases.

The author points out that to regard the genesis of the ulcer entirely on a venous basis is false. In practically all of his cases, arterial changes were demonstrated. The decreased supply of blood to the capillary bed and resulting lack of tissue nutrition is the major factor contributing to ulcer formation. Some cases have been observed in which trauma may have played a part.

Nineteen roentgenograms; 1 drawing; 1 table.

E. W. SPACKMAN, M.D.
Fort Worth, Texas

Significance of the Buckled Innominate Artery. Edward I. Honig, William Dubilier, Jr., and Israel Steinberg. Ann. Int. Med. **39:** 74–80, July 1953.

Buckling of the innominate artery may produce a prominent right superior mediastinal shadow on a con-

ventional postero-anterior chest film, which may be confused with a pathological process in this region, as aneurysm of the innominate or carotid artery, superior mediastinal tumor, retrosternal thyroid, lymph node enlargement, aneurysm of the aorta, or disease of the apex of the right lung. The phenomenon is usually associated with hypertensive and/or arteriosclerotic disease of the aorta. It is benign and is of no prognostic significance. Clinically the buckled vessel may be seen, felt, or auscultated. Definite diagnosis is made by angiocardiography.

Four roentgenograms; 4 drawings.

PAUL MASSIK, M.D.
Quincy, Mass.

TECHNIC AND APPARATUS

Orthoroentgenography. G. Schaltenbrand. Am. J. Roentgenol. **70:** 114–118, July 1953.

The author describes a method by which roentgenograms may be obtained which do not vary more than 0.5 mm. in size from the actual dimensions of the anatomic structure whose image is reproduced.

A screen containing a slit is placed between the object and the tube. During the period of exposure, the tube moves across the object in a direction perpendicular to the direction of the screen slit. The image thus obtained is exact in the direction of movement of the tube, with no distortion of size, but is exaggerated in the direction of the slit. An apparatus has been constructed which permits the tube to be moved by motor behind a slit in both the horizontal and vertical directions.

The author has used this procedure of roentgen measurement for the skull but envisions its usefulness for other parts of the skeleton as well, particularly measurements of the pelvis in obstetrical cases.

Nine roentgenograms; 5 photographs.

DORIS E. PIPKIN, M.D.
University of Louisville

A Simple Centering Device for Radiographic Tubes. Ove Mattsson. Acta radiol. **40:** 27–34, July 1953.

The author describes a simple optical system for localizing the center of the beam from a radiographic tube as well as indicating field size. This system consists of a tubular attachment mounted on the tube hood, containing a light and lens system which focuses the visible beam on a mirror mounted at an angle of 45°, the center of the mirror being in line with the central ray. A second light is included, which may be moved between the lens system and the mirror for the purpose of defining field size. The system utilizes a 10-volt circuit.

Five illustrations.

H. WENDELL WARD, M.D.
University of Arkansas

Multifilm Cassette for Use in Laminagraphy. Waldron M. Sennott and Howard E. Worrell. Am. J. Roentgenol. **70:** 141–142, July 1953.

The authors describe a multifilm cassette for laminagraphy consisting of a standard cassette reassembled with a lead-lined box frame of appropriate size, in which are placed four air-containing film spacers, each 1 cm. thick. To these film spacers are attached intensifying screens of varying speeds. The spacers are constructed by stretching airtight rubber dam over a balsa wood frame and inserting a small ball valve into an inside corner to provide for air refills. The advantage of this type of film spacer is that it decreases the absorption of radiation and provides a positive pressure film-screen contact. The multifilm cassette is easily adapted to a roentgenographic table of standard make without need of removing the Potter-Bucky diaphragm.

Five roentgenograms; 4 photographs.

CLAUDE D. BAKER, M.D.
Louisville General Hospital

MISCELLANEOUS

Hydatid Disease: Part I. W. J. Latham. J. Fac. Radiologists **5:** 65–81, July 1953.

In this excellent paper the author discusses the parasitology of hydatid disease and describes in detail its radiological aspects in the liver and lungs. Involvement of other organs is to be dealt with in another article.

There are three distinct types of hydatid disease: (1) the benign unilocular type, (2) osseous type, and (3) malignant alveolar type. The benign unilocular type of cyst has a two-layered, laminated wall. The inner, which is germinal, contains the cyst fluid and produces the brood capsules; the outer or ectocyst layer separates the cyst from the adventitia, which is the protective layer provided by the host. Apart from a trivial degree of toxemia and the dangers attendant upon rupture, the cyst manifests itself only by its increasing size and the pressure it exerts upon adjacent vital parts. Calcification is a common ending, but while this indicates a quiescent cyst, it does not necessarily mean that it is dead. Rupture is potentially accompanied by an infinite variety of disasters, dependent upon location, which are fundamentally variants upon the theme of secondary spread. When the adventitia of the cyst is damaged, it may become infected. The formation of daughter cysts is thought to be a protective reaction to trauma on the part of the hydatid.

The osseous type of cyst develops characteristics due to the narrow confines of its environment. The cyst continues to grow, as no protective adventitia is laid down and the process is silent for years. When the bone is eroded sufficiently to give way, the disease makes itself known.

The malignant alveolar type of disease is apparently due to a germinal layer which is not intact and so allows spread along the tissue planes and metastasis by way of the blood stream. The outcome, though delayed, is none the less fatal.

Unruptured cysts of the liver, if calcified, are usually not difficult to diagnose radiologically. Round, reticulated shadows, often multiple, are almost pathognomonic of hydatidosis. The uncalcified cyst is less easily recognized. It may project down from the edge of the liver, displacing adjacent viscera, or upward, producing an elevated diaphragm.

Infected cysts in the liver, as in the lung, may contain air, and the sign of the "camelotte" may be present. This is the shadow produced by the deflated cyst membrane floating upon the remaining fluid. In the chest, the infected cyst will nearly always produce pleural effusion or pneumonitis.

Rupture of a cyst of the liver into the bile passages may result in a non-functioning gallbladder and may be demonstrated by cholangiography. Pieces of hydatid membrane may be identified in the stool in patients in whom the cyst communicates with the intestinal

tract. Rupture into the peritoneal cavity may cause an anaphylactic reaction, hemorrhage, heterotopic cyst formation, or some form of peritonitis. A tremendous amount of fluid may form and a wide bore cannula must be used at paracentesis so that it will not be blocked by bits of hydatid membrane.

Pulmonary cysts may be primary or secondary, single or multiple, and peripheral or hilar in situation. Primary cysts account for 4 to 14 per cent of the total of hydatids. Secondary cysts are usually implanted by spread from beneath the diaphragm, but rarely may arise from a primary cardiac cyst or spread from a leaking primary lesion. Multiple cysts are not uncommon, but daughter cysts are rare in the individual lesion. The Escudero-Nemenov sign, change in the shape of the cystic mass during respiration, is a useful fluoroscopic finding. The majority of cysts are silent but may become symptomatic when rupture occurs. The coughing up of "grape-skins" is pathognomonic and occasionally a complete natural cure may be effected by this method. Rupture into the pleural cavity may be followed by hydropneumothorax, pyopneumothorax, or secondary multiple echinococcosis. Calcification is rare in lung cysts. Three characteristic x-ray appearances are produced when the adventitia communicates with the bronchial tree. The "sign of detachment" is due to air between the membranes. The "double arc sign" is found when some of the fluid is expectorated and there is seen an air/fluid level inside the cyst, which itself is surrounded by radiolucent air (the detachment sign). This indicates that the cyst membranes are no longer intact and its dissolution is certain. The "sign of the camelotte" is seen before the cyst cavity is obliterated, as the adventitial wall does not allow it to collapse immediately.

The author notes three rules which should be followed: never aspirate an hydatid (except possibly in bone), never perform a diagnostic pneumothorax if the mass being studied could possibly be a subpleural hydatid, and never perform formalage (replacing cyst fluid with formalin).

Twenty-seven roentgenograms; 2 diagrams; 1 photograph.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Acanthosis Nigricans. W. Roscher. Strahlentherapie **91**: 423–428, 1953. (In German)

The author differentiates between the benign and malignant form of acanthosis nigricans. The benign form is seen early in life, up to twenty years of age, is familial in occurrence, and associated with endocrine and developmental disturbances such as obesity, diabetes, and dysmenorrhea. The malignant form occurs usually after forty and frequently in association with a carcinoma elsewhere, especially in the abdomen, as carcinoma of the stomach in males and carcinoma of the uterus in females. The white and yellow races are predominantly affected; only two cases have been described in Negroes.

The author's patient was a male, aged 68, who had undergone radical excision of carcinoma of the lip. Within nine months there was a local recurrence, associated with acanthosis nigricans involving the axillae, both arms, and the head, as well as fatigue and a dry cough. Repeated gastrointestinal examinations were done because of low hemoglobin (28 per cent) and occult blood in the stools. Finally a roentgen diagnosis of carcinoma of the cardia was established.

One roentgenogram; 2 photographs.

ERNEST KRAFT, M.D.
Newington, Conn.

Discussion on Scleroderma. Edward R. Cullinan, R. A. Kemp Harper, *et al.* Proc. Roy. Soc. Med. **46**: 507–522, July 1953.

Cullinan opens this symposium on scleroderma with a rather general clinical discussion, including 4 brief case histories to illustrate the course. The syndrome is characterized by sclerosis of the dermis of the hands with possible involvement of the forearms, upper trunk, and face, and occasionally the feet and legs. Associated changes may occur in the gastrointestinal tract. Skin changes may be accompanied by Raynaud's phenomenon, phalangeal absorption, pigmentation, telangiectases and calcinosis. The condition is confined almost wholly to women.

Kemp Harper reports a series of 14 cases. He describes the soft-tissue and bony lesions, the changes in the lungs and heart, and at greater length the alterations in the alimentary tract.

Radiographic evidence of scleroderma is seen in the fingers as calcification on the palmar aspect of the terminal phalanges. It may also occur in the hands, elbows, buttocks, shoulders, and legs. Calcification may occur in the soft tissue without overlying skin changes visible clinically. Bone changes consist of absorption of the tufts of the terminal phalanges from decreased blood supply.

Lung changes, producing dyspnea, are a combination of fibrosis and cyst formation. Cardiac enlargement may occur from involvement of the heart muscle by the degenerative process and from increased pulmonary resistance to blood flow.

In the esophagus, diminution or absence of peristalsis is the outstanding feature. This is demonstrable only in the recumbent position. In severe cases there is pooling of barium in the lower half of the esophagus. There may be localized narrowing at the lower end, with proximal dilatation.

A decrease of motility in the small intestine and the development of diverticula (where the muscular coat disappears in a patchy manner) are seen. In severe cases, barium may remain in the small intestine for forty-eight hours, producing symptoms of obstruction. In these cases surgical relief is a very temporary palliative measure.

The colon is often affected. Large diverticula form and simulate haustra except that striking asymmetry occurs, so that a wide-mouthed diverticulum may be opposite a haustral indentation. Furthermore, after evacuation of a tannic-acid enema, the diverticula remain uncontracted, while the rest of the bowel contracts well. There may be no symptoms, but with marked changes there is usually a history of alternating constipation and diarrhea. Occasionally the descending colon may show changes suggestive of ulcerative colitis. A typical, gray, sclerodermatous mucosa is seen on sigmoidoscopy.

The pathological changes in the intestinal tract are loss of muscle, thickening of the submucosa, and infiltration with leukocytes and plasma cells, with hypertrophy of the connective tissue.

Brief comments by others complete the symposium.

Thirty-two roentgenograms; 4 photographs.

D. DE F. BAUER, M.D.
Coos Bay, Ore.

Pediatric Radiology. Edward B. D. Neuhauser and Martin H. Wittenborg. New England J. Med. **249:** 62–68, July 9, 1953.

As the authors state in their conclusions "No field of diagnosis is expanding more rapidly than pediatric roentgenology. Satisfactory roentgenologic criteria for the diagnosis of well known conditions are being established, new x-ray signs are being disclosed, and many diseases formerly thought to be rare are being recognized with increasing frequency."

In this paper recent contributions to this important field are reviewed, and a bibliography is appended. Most of the papers cited have appeared in RADIOLOGY either as original contributions or in abstract form.

RADIOTHERAPY

Carcinoma of the Oral Cavity and the Lower Jaw. S. P. Srivastava. J. Indian M. A. **22:** 450–456, August 1953.

In India, Ceylon, and the Philippines, carcinoma of the oral cavity, especially the cheek, is the most common type of malignant lesion, because of the practice of chewing either betel nuts or tobacco mixed with lime. The author states that most cases seen are too far advanced for radium implantation or surgery and consequently are treated with roentgen rays. He gives 3,000 to 4,000 r (tumor dose) with 200 kv. in two to three weeks in an attempt at palliation.

Surgical treatment of oral cancer arising at different sites is discussed, but no results are given for either surgery or irradiation. ZAC F. ENDRESS, M.D.
Pontiac, Mich.

Radiation Treatment of Hemangiomas. Johannes Thoms and Niels Fjeldborg. Acta radiol. **40:** 39–53, July 1953.

For evaluation purposes, cases of hemangioma first treated in 1941 were used in this series. The study is based upon 247 patients with 293 hemangiomas seen at the Radium Center in Aarhus, Denmark. The stellar hemangiomas (spider marks) were excluded, most of them being treated by electrodesiccation. The method of treatment varied according to the size and type of the lesion and effect obtained. Contact therapy by roentgen rays (60 kv.) or radium filtered with 0.1 mm. of monel metal was used in most instances, or a combination of these.

Most capillary hemangiomas were treated with radium or Bucky rays (12 kv.). A few disappeared spontaneously, without treatment. The superficial cavernous hemangiomas were given contact radium therapy, for the most part alone, but in a small percentage of cases in combination with short-distance roentgen therapy. Mixed types were mostly given soft radiation alone or in combination with radium.

The results were classified as "excellent" if the lesion disappeared and the skin became normal. A satisfactory cosmetic result with soft normal skin consistency was classified as "good," though the site of the hemangioma could still be recognized. Of 34 cases of capillary hemangioma, a good result was obtained in 7; 17 cases were unchanged; in 9 there was residual marginal hemangioma, and in 1 an unsightly scar, with pigmentation. In 80 per cent of 182 cases of superficial cavernous hemangioma, an excellent or good result was achieved. Excellent results were also obtained in 80 per cent of the 43 cases of deep cavernous and mixed-type hemangiomas.

A follow-up on 60 patients, carried out ten years or more after treatment was completed, showed examples of both improvement and deterioration of the immediate result.

Six case histories are included, with 18 illustrations; 3 tables. GEORGE REGNIER, M.D.
University of Arkansas

Radiation Therapy of Hemangioma. P. Hess and B. Fischer. Strahlentherapie **91:** 256–260, 1953. (In German)

The authors report on 911 cases of hemangioma encountered in an experience of more than twenty years. There were 624 females and 287 males. Five hundred and seventy-five cases are evaluated as to results.

Three methods of radiation treatment were used. Grenz rays were given in cases of hemangioma simplex, in dosages of 1,600 to 2,000 r in air, four to six weeks apart, up to a total dose of 10,000 r. In children under two years of age, 87.8 per cent perfect cosmetic results were obtained. Contact radiation and radium moulages were used for the treatment of cavernous hemangioma. With the Chaoul tube, 400 r in air were delivered per sitting, once a month, with an average total dose of 1,600 r; 86.6 per cent good results are reported. With radium moulages, 600 gamma r were given in one sitting and the dose was repeated three to four months later. The average total was 1,200 gamma r. Good results were obtained in 90.6 per cent.

Even though spontaneous regression of hemangioma is observed, the authors believe in early radiation therapy, because of the marked decrease of radiosensitivity with increase in age. They prefer contact radiation because of its simplicity of application.

Three tables. EUGENE F. LUTTERBECK, M.D.
Cook County Hospital, Chicago

Hemangioendothelioma of the Vagina. Abraham Marck, Carlton Wirthwein, and Abraham Melamed. Am. J. Obst. & Gynec. **66:** 436–440, August 1953.

Hemangioendothelioma is a rare malignant neoplasm of vascular origin, with a definite and often marked tendency to hemorrhage. Metastasis may occur through both vascular and lymphatic channels.

A case is reported of an 18-year-old patient who was seen first for intermenstrual bleeding. A sessile mass with an ulcerated purplish surface was found in the vaginal fornix. This was excised and reported as hemangioendothelioma. Seven months later a recurrence was excised and coagulated. There was no evidence of reappearance of the tumor after an interval of two years. Eight months later, however, post-coital bleeding occurred, and the cervix was found to be displaced posteriorly and hidden by a mass. The lateral vaginal fornices appeared to contain deposits of tumor tissue. External x-ray therapy was given anteriorly and posteriorly for a tumor dose of 4,000 r. The tumor was very radiosensitive and pelvic examination two months after completion of therapy was normal. Vaginal bleeding recurred fourteen months later. Surgical exploration showed a mass in the left pelvis, extending into the left

broad ligament and anterior fossa and adherent to the sigmoid colon. Pulmonary metastases were subsequently demonstrated. Death occurred some four and a half years following the first symptoms.

In view of the tendency of this tumor toward recurrence following local excision, the authors conclude that radical extirpation by surgery or irradiation or a combination of these two should be considered in the treatment.

Two roentgenograms; 1 photograph; 1 photomicrograph.

MASON WHITMORE, M.D.
Jefferson Medical College

Malignant Lymphomata. A Review of the Pathology, Clinic Features and Therapy. Axel Scheel and Eivind Myhre. Acta radiol. **40:** 63–80, July 1953.

Having reviewed the diverse terminology applied to the malignant lymphomas, the authors undertook to determine to what extent group classification is of practical clinical significance and whether the various groups demonstrate characteristic clinical features, such as variations in radiosensitivity or in prognosis. They studied 134 cases of malignant lymphoma, which were classified as (1) lymphogranulomatosis (Hodgkin's disease); (2) reticulosarcoma; (3) lymphosarcoma; (4) follicular lymphoma. They made no attempt to determine whether one type may show transition to another.

There were 49 cases of reticulosarcoma, 41 cases of lymphosarcoma, 3 cases of follicular lymphoma, and 41 cases of lymphogranulomatosis. The lymphosarcomas were subdivided into lymphoblastoma, 16 cases, and lymphocytoma, 25 cases.

The material presented includes all patients with these diagnoses who received treatment at the Radium Hospital, Oslo, during the period 1936–47. All data were compiled as from the day of admission, with no consideration of the duration of the disease before that time. The age distribution varied considerably. There were twice as many males as females.

In a tabular presentation of the regions involved at the beginning of treatment, it is noted that lymphogranulomatosis involves one lymph node region in just under one third of the cases. Lymphosarcoma was somewhat more frequently limited to one region, but when it extended to more than one area, several were usually affected. About one fourth of the cases of reticulosarcoma involved a single region. It is of particular note that the lymphocytomas occurred equally frequently in one lymph node region and in several, and in that respect perhaps have a better prognosis than the lymphoblastomas.

In lymphogranulomatosis involvement is most frequently intrathoracic, mediastinal tumors being predominant. In lymphosarcoma and reticulosarcoma there is a greater incidence of involvement of organic systems other than those of the chest, as the skeleton and abdomen. In about a third of the patients with lymphosarcoma there was a significant increase in the white-cell count. All of these patients with leukocytosis had a considerable spread of the disease and all died in the course of two years.

All the patients in the present material were treated with irradiation. In a small number of cases surgical extirpation was first attempted. The irradiation was of two categories: (1) a minimum skin dose of 3,000 roentgens over the lesion and (2) lesser doses of irradiation. Deep therapy technic was employed throughout. Twenty-eight per cent of the total of 134 patients were alive after five years. The highest rate of survival was in the lymphogranulomatosis group, the lowest in reticulosarcoma. There was an even distribution of fatalities over the five years with lymphogranulomatosis, while in lymphosarcoma and reticulosarcoma the tendency was to a high incidence of deaths in the first year. All patients with lymphosarcoma and reticulosarcoma who survived five years were alive and symptom-free at the time of this report. More than 80 per cent of the patients with lymphosarcoma and reticulosarcoma either succumbed in the course of the first year or survived free from recurrence during the whole of the period of observation. The prognosis in lymphoblastoma is very poor; it is considerably better in lymphocytoma.

It is further justifiable to conclude that the irradiation dose is of significance to the prognosis, with the proviso, however, that the disease is not too widespread. In those patients in whom a complete course of irradiation was possible with the larger dose, a better statistical survival was obtained. The authors believe that the significance of the conception of acquired radioresistance may possibly have been somewhat exaggerated in the past.

Other factors are evaluated, but these appear to be of little significance.

Twelve photomicrographs; 7 tables.

I. MESCHAN, M.D.
University of Arkansas

Cardiac Involvement in Malignant Lymphoma. J. D. N. Nabarro. Arch. Int. Med. **92:** 258–264, August 1953.

Although pathological evidence of cardiac involvement is often found in cases of malignant lymphoma, corresponding clinical manifestations are uncommon and, when they do occur, usually give rise to considerable diagnostic difficulty. The nature of the diagnostic problem varies with the stage in the development of the lymphoma at which the cardiac symptoms appear. In the rare cases in which the early or presenting symptoms are those of heart disease, the correct diagnosis has seldom been made before autopsy. When a patient known to have a malignant lymphoma exhibits cardiac complaints, their neoplastic origin may be recognized, but in older patients they may be attributed to coronary disease. In the terminal stages, anemia and obstructive symptoms and signs often produce a picture closely resembling congestive heart failure, and the presence of cardiac involvement may be overlooked. Treatment by irradiation has proved disappointing.

Three cases are reported to illustrate some of the clinical manifestations that may result from cardiac involvement in malignant lymphoma: Case I, congestive heart failure as the presenting symptom of reticulosarcoma; Case II, pericarditis occurring in the course of Hodgkin's disease; Case III, Hodgkins' disease with terminal congestive cardiac failure. In Case I the diagnosis was made by biopsy of a skin nodule. The tumor was highly radiosensitive, but treatment was unsuccessful.

Two roentgenograms; 2 photomicrographs.

Radiation Therapy of Cancers of the Cervix from 1931–1946. N. Nicolov. Strahlentherapie **91:** 161–193, 1953. (In German)

The results of radiation therapy in 1,452 cases of

cancer of the cervix, treated from 1931 to 1946 in the City Hospital of Wien-Lainz, are reported. The following method was used:

1. Radium, 80 mg., intravaginally for thirty hours.
2. Two weeks later, 80 mg. radium intravaginally for twenty-four hours.
3. Two weeks later, 40 mg. radium within the uterus for seventy-two hours.
4. Four weeks later, 1,600 r tumor dose to the right and left parametria, with deep roentgen therapy or a teleradium source.

The vaginal applicator contained 6 tubes with 13.33 mg. radium each, filtered with 1.7 mm. platinum. The uterine applicator was a 7-cm. long wire cylinder, containing 3 tubes of 13.33 mg. radium with 1.5 mm. platinum filtration.

From 1931 to 1946, 1,960 cases were seen: 97, or 5 per cent, Stage I; 222, or 11.3 per cent, Stage II; 1,175, or 60 per cent, Stage III; 466, or 23.7 per cent, Stage IV.

The following results were obtained in 1,452 cases. *Stage I*, 94 cases, 72 alive without symptoms after five years, a relative cure rate of 76.6 per cent; *Stage II*, 213 cases, 116 alive after five years, a relative cure rate of 54.5 per cent; Stage III, 862 cases, 223 alive after five years, 25.8 per cent relative cure rate. No patient with Stage IV disease lived for five years.

Radium damage was observed in 209 cases, or 14.3 per cent of the entire series. In 136 cases, or 9.3 per cent, the lesions were reversible, including proctitis, cystitis, and cervicitis; 73 patients, or 5 per cent, had irreversible changes. Thirty-six patients, 2.5 per cent of the total, had fistulas.

Most of the recurrences were seen after one and two years, some up to thirteen years and later. There were 314 recurrences among 725 primarily cured patients. Most of these were treated with a second series of teleradium therapy.

The author comes to the conclusion that an average of 40 per cent of cases of cancer of the cervix can be cured with radiation therapy, a result which is superior to that afforded by surgical treatment.

One photograph; 12 tables.

EUGENE F. LUTTERBECK, M.D.
Cook County Hospital, Chicago

Roentgen Therapy of Female Beard. E. H. Graul. Strahlentherapie **91:** 405–409, 1953. (In German)

Although radiotherapy for hypertrichosis has been condemned for decades, the author tried contact x-ray therapy on five patients in 1949 and 1950 following encouraging results reported by Knierer (Strahlentherapie **79:** 257, 1949). In four to six months the cosmetic results were excellent, although minimal skin atrophy and slight loss of pigmentation were noted. After two and three years, however, atrophic scarring with orange-peel appearance, telangiectases, and depigmentation developed. The author considers the results disappointing and warns emphatically against permanent x-ray epilation.

Four photographs. ERNEST KRAFT, M.D.
Newington, Conn.

Treatment of Plantar Warts. Rud. Koch. Strahlentherapie **91:** 416–422, 1953. (In German)

One-hundred and twenty-three patients with 222 plantar warts were treated by irradiation between 1941 and 1952. The sexes were equally represented. The predominant incidence was in the second decade of life. Through 1949 radium was used, 79 cases being thus treated, with cure in 94 per cent. In the cases seen from 1950 on, contact roentgen therapy (Chaoul) was employed, with 98 per cent cures and a single recurrence. One treatment of 1,000 r was given at 60 kv., 4 ma., occasionally supplemented by an additional 500 r after three weeks.

The chief advantage of contact roentgen irradiation over radium therapy lies in the possibility of administering a single massive dose in a minute's time, while radium requires slow application with repeated doses.

Two photomicrographs; 2 graphs; 7 tables.

ERNEST KRAFT, M.D.
Newington, Conn.

RADIOISOTOPES

Diagnosis of Breast Cancer with Radioactive Phosphorus P^{32}. K. L. Bhattacharya, R. Datta-Chaudhury, A. Bose, and N. N. Das-Gupta. J. Indian M. A. **22:** 393–396, July 1953.

The authors used radioactive phosphorus preoperatively to differentiate benign from malignant tumors of the breast. This is possible because rapidly growing cells have a higher metabolic rate and need increased amounts of phosphorus for nucleo-protein formation. A single dose of 500 μc. of P^{32} was given intravenously and measurements, with a Geiger-Müller counter, were made of surface radioactivity over suspected and normal sites at two, six, twelve, twenty-four, and forty-eight hours after the injection. Since P^{32} emits beta particles which do not travel more than 8 mm. through tissue, only tumors near the surface can be detected by this method.

Fifteen cases were studied. Seven cases of benign tumors had measured surface activities less than 140 per cent as compared to normal breast tissue. In 7 cases of duct carcinoma the surface activity over the tumor exceeded 140 per cent. In one case of duct carcinoma, however, the activity was only 105 per cent. This may have been due to a deep-lying tumor. In 2 cases of cancer with ulceration and infection the readings were over 300 per cent. Measurements over the axillary and supraclavicular metastatic nodes showed a surface activity of more than 140 per cent of normal.

Two illustrations; 3 tables. PAUL MASSIK, M.D.
Quincy, Mass.

Diagnosis of Thyrotoxicosis by a Simple Out-Patient Radioactive Iodine Technique. Alastair G. Macgregor, H. Miller, P. J. Blaney, and W. S. Whimster. Brit. M. J. **2:** 21–22, July 4, 1953.

Function of the thyroid gland as evidenced by ability to absorb iodine has been assessed by direct measurement of the amount or rate of uptake in the patient's neck or by measuring the amount excreted in the urine, the latter indicating the remainder of the administered dose not utilized by the thyroid. The capacity of the thyroid gland to synthesize and discharge thyroid hormone can also be measured by assaying the radioactivity present in the blood plasma.

The authors determined the radioactivity present in the protein-bound fraction of the plasma forty-eight hours after the administration of a tracer dose of 25 to 30 μc. as studied in 50 patients. All of these patients were followed and studied to the eventual conclusion of a positive diagnosis. In 28 patients the results of the tests and the clinical assessment were in full agreement, and in a further 19 patients a doubtful clinical diagnosis was confirmed or an erroneous one corrected. In 3 cases misleading information was provided by the assay of the blood plasma.

The results of the test were interpreted by the authors as follows. Levels of activity in excess of 0.4 per cent per liter in the protein-bound fraction were considered to indicate definite thyrotoxicosis; values below 0.2 per cent per liter were interpreted as excluding the presence of thyrotoxicosis. Intermediate values, lying between 0.2 and 0.4 per cent per liter, were interpreted according to the proportion of the whole plasma activity that was protein-bound, proportions over 50 per cent usually indicating thyrotoxicosis even if the absolute amount in the protein-bound fraction was below 0.4 per cent per liter, though accuracy of measurements below this level of activity is not great.

In addition to the 50 persons under study, samples from 22 normal persons who were definitely non-toxic were also analyzed. In each instance the correct interpretation was made on the test of the blood plasma.

One table.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Criteria for Therapy of Malignant Thyroid Lesions with I^{131}. Charles V. Meckstroth and George M. Curtis. Arch. Surg. **67**: 187–193, August 1953.

The authors report their experience with 47 consecutive cases of thyroid carcinoma of three types: 13 cases of papillary carcinoma, 17 of adenocarcinoma with varying amounts of colloid formation, and 17 cases of undifferentiated carcinoma. None of the undifferentiated or papillary types showed appreciable uptake of I^{131} in the primary or metastatic lesions. Eight of the 17 patients with adenocarcinoma had metastases, and 3 of these showed sufficient uptake to indicate I^{131} therapy. These 3 cases are reported. All the patients were benefited by the treatment.

While the major indication for radioactive iodine therapy is the presence of functioning inoperable metastases, there are two other concrete indications: (1) to destroy the normally functioning thyroid tissue which may remain, whereupon the thyroid-stimulating hormone of the anterior pituitary may encourage the metastatic deposits to take up and retain a significant amount of I^{131}; (2) on occasion, to make possible a study of the distribution of uptake in surgical or autopsy specimens.

Other pertinent observations made following clinical research are (1) that radioactive iodine is of little help in diagnosing carcinoma in a patient with nodular goiter without metastases; (2) that in patients with metastases from an unknown source, the failure of I^{131} to localize in these lesions does not rule out the possibility of their being of thyroid origin. [This last is not what the authors say but is obviously what they mean. —Ed.]

Five illustrations, including 2 roentgenograms and 1 radioautograph.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Experiences in Endovesical Irradiation of Bladder Tumors with Liquid Radioactive Cobalt. Walter Bessler. Strahlentherapie **91**: 451–459, 1953. (In German)

Thirty cases of bladder tumor were treated with an aqueous solution of cobalt 60, emitting 96 per cent gamma rays and 4 per cent beta rays during disintegration. Sixty cubic centimeters of the isotope solution were injected into the balloon of a Bardex catheter, which had been inserted into the bladder. This volume has a strength of 18 to 25 mc. Co^{60} and produces 800 to 1,300 r in twenty-four hours on the surface of the balloon. The total dose was 10,000 to 12,000 r within eight to ten days. When surgery was performed, a prophylactic postoperative dose of only 8,000 to 10,000 r was applied.

For malignant papilloma and superficial carcinoma the isotope therapy was found to be the treatment of choice. There was no recurrence in a series of 13 cases. Good results were also obtained in recurrent benign papillomas. For infiltrating carcinoma, surgery is advised or, if the growth is inoperable, either betatron or supervoltage irradiation, isotope therapy being inefficient in this type of involvement.

One roentgenogram, 3 tables.

ERNEST KRAFT, M.D.
Newington, Conn.

Experimental Observations on Local Tumor Therapy with Radiogold. Helmut Richard Kanitz, Friedrich Pfander, and Hanno Poppe. Strahlentherapie **91**: 208–222, 1953. (In German).

If colloidal radioactive gold is injected into a solid tumor, about 6 to 10 per cent is carried into the distant tissues. The authors report a method in which such loss is decreased by 90 per cent.

The maximum intensity of available radiogold in Germany is 780 mc. per gram as compared with 3,000 mc. per gram in the United States. Goldsol has a concentration of 1.8 to 2.3 mc. per c.c. as compared with 30 mc. per c.c. in the United States. Large amounts of fluid are therefore required to obtain high concentrations, and in many cases this is undesirable.

Experiments were done on 60 guinea-pigs and 7 patients. It was shown that after injection of 0.4 c.c. of 1 per cent stable $AuCl_3$ into the muscles of an animal, followed by the same amount of *l*-ascorbic acid, the metallic gold was precipitated and remained in the area for twelve months, as shown radiographically. The same was accomplished with Au^{199}. When the amounts of gold found in different organs after injection of colloidal gold, with and without the reduction method, were compared, the following results were obtained:

	Colloidal Gold without Reduction Method (%)	Colloidal Gold with Reduction Method (%)
Liver	6.94	0.29
Spleen	10.94	0.91
Kidney	3.87	0.89
Intestine	8.07	0.83
Vertebra	1.8	0.43

In 7 patients, the sodium ascorbic acid was injected before, during, and after the administration of radiogold. It remained in the injected area and could be recognized as a bluish deposit through the skin.

The authors believe that the method warrants further

trials, particularly in lesions difficult of access, as in the hypopharynx.

The technic of compounding gold chloride and colloidal gold, the examination of organs and tissues, and the measurement technics are described in detail.

Five photographs; 5 tables.

EUGENE F. LUTTERBECK, M.D.
Cook County Hospital

Localization of Radioactivity in Regional Lymph Nodes. Harold F. Berg, William M. Christophersen, Avrom M. Isaacs, and J. Ray Bryant. Arch. Surg. **67**: 228–242, August 1953.

The authors describe experimental work on apparently healthy mongrel dogs following the injection of varying amounts of radioactive gold, Au^{199}. In one group of dogs the injection was made through a ureteral catheter placed in a segmental bronchus. The gold was administered in doses of 2 to 5 mc. in 5 c.c. of isotonic sodium chloride solution. Amounts ranging from 3 to 35 mc. were instilled in this manner. In another group of dogs the isotope was introduced by way of a long needle passed through a bronchoscope into the mucosal wall of the right intermediate bronchus; 2 mc. in a 2 c.c. volume was injected in each animal. In still another group of dogs 5 mc. of Au^{199} in 5 c.c. of saline solution, to which 300 turbidity units of hyaluronidase had been added, was injected into the muscular layer of the urinary bladder at the fundus.

Assay of the dogs receiving the radioactive gold by the instillation method and killed at various intervals, revealed localization of activity within the selected segments of lung and regional lymph nodes. Histologic studies showed radionecrosis in proportion to the amount of isotope instilled in the segments of lung. Only minor changes were observed in lymph nodes. When the Au^{199} had been injected directly into the mucosa of the intermediate bronchus, assay revealed extremely high levels of concentration of radioactivity in the regional lymph nodes, a lower level at the site of injection, and negligible activity in remote organs such as liver and spleen. In the urinary bladder, concentrations of radioactivity were found to be consistently high at the site of the injection, as well as in the trigone and in the regional lymph nodes. Necrosis at the site of injection was observed.

The authors point out that this work was done on healthy dogs, without carcinoma, and suggest that it is possible that localization of radiogold in lymph nodes would not occur in the presence of carcinoma if the lymphatics were "plugged." Clinical application and trial on human beings with cancer is suggested as the next step in determining the possible value of radioactive isotopes injected in this manner.

Seven photomicrographs; 4 photographs; 2 drawings; 4 tables.

RICHARD A. ELMER, M.D.
Atlanta, Ga.

Studies on Blood-Brain Barrier with Radioactive Phosphorus. III. Embryonic Development of the Barrier. Louis Bakay. Arch. Neurol. & Psychiat. **70**: 30–39, July 1953.

The concentration of P^{32} by the brain in rabbits varies inversely with age from the early intra-uterine period to seven weeks after birth, at which time the concentration is equal to that in the adult brain. This was demonstrated by injecting each of 14 rabbits, of which 5 were pregnant, with a single dose of P^{32}, ranging from 100 to 250 μc., twenty-four hours before the animals were killed and then studying the brains for radioactivity by scalers, and in a few cases by radioautographs. The animals were killed at various stages of life. Both the rabbits and fetuses were studied.

The results suggest that there is a blood-brain barrier which becomes less permeable with increasing maturity. The P^{32} in the adult brain is 20 to 40 per cent of the amount in adult blood. Other organs fail to exhibit this change in blood-organ permeability but have a generally higher phosphate concentration than the brain.

A discussion of permeability studies by other workers is included.

Six radioautographs; 3 graphs; 2 tables.

PAUL MASSIK, M.D.
Quincy, Mass.

Autoradiography of Mast Cells in Experimental Skin Tumors of Mice Injected with Radioactive Sulfur (S^{35}). G. Asboe-Hansen. Cancer Res. **13**: 587–589, August 1953.

Previous histochemical and chemical studies have indicated that the granules of the connective-tissue mast cells contain a sulfuric mucopolysaccharide. This substance is closely related to heparin and hyaluronic acid without being identical with either. These studies, however, reveal nothing concerning the location of sulfur in the tissue. The author therefore used autoradiography to demonstrate the localization of sulfur-containing compounds in connective tissue, their relative quantities, and distribution.

In order to obtain connective tissue containing mast cells in large numbers, experimental skin tumors (papillomas), containing cells presumably at the maximum of their functional capacity, were induced in mice by a single painting with 9,10-dimethyl-1,2-benzanthracene. Nine of the tumor-bearing mice received one intraperitoneal injection of S^{35}, 8 μc./gm. of body weight with 0.1 mg. Na_2SO_4 as carrier in 0.2 ml. of sterile water. After forty-eight hours the mice were killed and the tumor and a piece of normal skin from a symmetrical site were excised. Autoradiographs were made, applying the stripping film technic. These studies showed that the majority of mast cells take up sulfur. This uptake manifests itself as a blackening of a stripping film. Whether the ability of mast cells to take up radiosulfur depends on their age and metabolic state, whether the experimental period (forty-eight hours) is too long or too short, whether or not the sulfuric acid content of individual mast cells varies—these are problems which require extensive investigations. Such studies are in progress.

Four radioautographs.

Chloride "Space" and Total Exchanging Chloride in Man Measured with Long-Life Radiochloride, Cl^{36}. S. A. Threefoot, G. E. Burch, and C. T. Ray. J. Lab. & Clin. Med. **42**: 16–33, July 1953.

Long-life radiochloride, Cl^{36}, was administered to 2 control subjects and 3 persons with congestive heart failure. Samples of blood and all excreta were assayed radiometrically daily for Cl^{36} and chemically for Cl^{35}, Na^{23}, and K^{39}, as well as volume of water. Intake of these substances was controlled for thirty to sixty days. From these measurements, the chloride "space" and "total" chloride mass were calculated by methods involving isotope dilution and biologic decay rates.

The space expressed either as liters serum equivalents

or as per cent body weight was larger in the subjects with congestive heart failure, being largest in those with edema and anasarca. The controls had a mean of all daily calculated space values of 34.8 and 22.5 per cent body weight and the patients with failure 36.1, 65.6, and 60.8 per cent body weight. The latter 2 individuals had severe congestive failure, uncompensated. The mean calculated "total" chloride mass was 59.0 and 40.4 gm. for the controls and 85.2 gm., 147 gm., and 88.7 gm. for the subjects with congestive heart failure. These results demonstrate that calculated values for "space" and "mass" show wide variations from day to day, which are more marked in the subjects with congestive failure than in the controls.

Isolated determination of space or total mass of chloride could be misleading. It is apparent that certain of the *calculated* values are not representative of the *actual* space and mass. This is especially true when there is a disturbance in the "steady state" and equilibrium of distribution of the tracer, produced by therapeutic procedures, changes in intake of the non-tracer, or changes in the state of disease. Examples of such instances are presented when variations in the body weight and calculated space are discordant. The same inadequacies observed with an isotopic tracer, plus biologic and chemical differences, are present when other substances such as bromide or thiocyanate are used to measure chloride space or when inulin, sucrose, thiosulfate, or dyes are employed for measurement of extracellular fluid space. There is also indirect evidence that significant quantities of chloride are distributed in cells, bone, or other sequestrated compartments.

Although the physiologic and quantitative significance of the discrepancies between the "calculated" and "probable actual" values is not known, these factors must be at least considered in the experimental determination of electrolyte spaces and masses by any tracer methods, and calculations based on the assumption that all chloride is extracellular must be regarded with skepticism.

Six charts; 5 tables.

Studies with Radioactive Colchicine. I. The Influence of Tumors on the Tissue Distribution of Radioactive Colchicine in Mice. A. Back and E. J. Walaszek. Cancer Res. **13**: 552–555, August 1953.

The distribution of radioactive colchicine, prepared by biosynthesis, was studied in normal and tumor-bearing mice. The spleens of normal mice accumulated significant amounts of injected radioactive colchicine (39–47 per cent of that recovered in the body), while none was found in the spleens of mice with tumors. The intestines of the tumor-bearing mice, however, accumulated more colchicine than the intestines of the normal animals.

When lyophilized tumor tissue was injected into normal mice, it had an action similar to that of a tumor *in situ*. It is postulated that the described effects in spleen and intestine are provoked by a substance or substances present in tumor tissue.

Four tables.

Effect of Priscoline on the Clearance of Radiosodium from Muscle and Skin of Man in Normal and Diseased Limbs. Jack Freund, Lawrence H. Wisham, and Rosalyn S. Yalow. Circulation **8**: 89–97, July 1953.

Clearance of radiosodium after injection into the skin of the calf and the gastrocnemius muscle was studied in normal subjects and in patients with evidence of peripheral vascular disease. A mica-window Geiger counter was used with appropriate scaling to provide the data. Each patient was then given 37.5 mg. of Priscoline into the femoral artery on the same side, followed in two or three minutes by radiosodium injections into the skin and muscle of the calf as before. Counting was carried out and the results compared with those obtained without Priscoline. Tables present the results of the experiments expressed both in terms of clearance half life and clearance constant.

It was found that there was a constant but very small increase in the rate of clearance of radiosodium from the muscle and a significant increase in clearance from the skin following administration of Priscoline.

It is concluded that, since the effect of Priscoline on local muscle flow is small and far less than the effect of moderate exercise, the value of this drug in relieving intermittent claudication in patients with organic occlusion is questionable. Its suggested use for maintaining the integrity of the skin in peripheral vascular disease appears to have some justification when the drug is administered intra-arterially.

Two graphs; 4 tables. ZAC F. ENDRESS, M.D.
Pontiac, Mich.

RADIATION EFFECTS

Brain Necrosis Following X-ray Therapy. Eldon L. Foltz, John B. Holyoke, and Henry L. Heyl. J. Neurosurg. **10**: 423–429, July 1953.

Although brain and nervous tissue are usually resistant to direct damage by x-ray radiations, there is increasing evidence that in rare instances the vascular system of the brain can be severely damaged by irradiation. The authors report the case of a 45-year-old female who received superficial x-ray therapy to a basal-cell epithelioma of the scalp. Two and a half years later, evidence of a progressing intracranial lesion developed. The original lesion was diagnosed by biopsy and 2,500 r (air) were administered to the right temporal scalp. A 5 × 5-cm. field, 25 cm. target-skin distance, inherent filter (1 mm. Al equivalent), 120 kv., 20 ma., and 15 min. 30 sec. time, were the factors of the single treatment. The skin dose was 2,925 r; at 2 cm. (approximately at the surface of the temporal lobe), the depth dose was 1,287 r; and at 5 cm. (the approximate maximum depth of operation), the depth dose was 526 r.

The patient presented signs of a progressing intracranial lesion and increasing intracranial pressure two years and a half folllowing the treatment. In the right temporal area of the scalp was a depilated area, 5 × 5 cm., of thin, atrophic, scarred skin with a central 1-cm. ulcer. A decompression was first done as a life-saving procedure. This was followed about a month later by a right temporal craniotomy. A right temporal lobe brain lesion was found immediately underlying the irradiation scar on the scalp. This proved to be a sharply limited area of ischemic infarction of the brain due to progressive, obliterative vascular disease. The main blood vessel changes observed were: (1) hyalinization and thickening of the wall, (2) suggestive amyloid

degeneration, and (3) telangiectasis of subpial vessels. The cerebral edema that accompanied the lesion was unexplained and was probably similar to that which is often seen with brain tumors.

The authors assume that the dosage was too great, in this particular patient, to be tolerated by the cerebral vascular system. They feel, however, that the vascular changes may have been the result of individual causes more subtle than the relatively simple factor of overdosage.

Four photomicrographs.

SEYMOUR A. KAUFMAN, M.D.
Boston, Mass.

Effect of Therapeutic Irradiation of Carcinoma of Cervix on Liver Function. J. R. Snavely, R. H. Bullington, and J. V. Schlosser. Arch. Int. Med. **92:** 195–203, August 1953.

Fifteen patients with carcinoma of the uterine cervix were studied serially with several standard liver function tests as they were being treated with x-rays and radium. The irradiation was given in accordance with the plan employed at Charity Hospital, New Orleans, consisting of 24 x-ray treatments to the pelvis in a period of about thirty days, followed by radium. The x-rays were administered through six fields, two anterior, two posterior, and two lateral, being directed perpendicularly so as to cross-fire the lateral edge of the parametrium. The following factors were employed: 400 kv., composite filter (Thoraeus) yielding a half-value layer of 5 mm. Cu, and a focus-skin distance of 50 cm. A precalculated tumor dose of 3,000 r was administered to the lateral edge of the parametrium in the region of standard reference point B, located 5 cm. from the mid-line at a level 2 cm. above the lateral fornix. Radium therapy was then given with a tandem in the cervical canal and Regaud type (Kaplan) colpostats containing radium in the vaginal vault. The quantities of radium and their distribution were varied to suit the anatomical dimensions and were calculated to deliver a constant dosage of 6,600 r to the paracervical triangle, standard reference point A, located 2 cm. from the mid-line at a level 2 cm. above the lateral fornix.

In 10 of the 15 patients, an increased retention of sulfobromophthalein (bromsulfalein), above control values, was found. This retention was significantly more often present in those whose total energy absorptions from ionizing radiations were high than in those with comparatively low energy absorptions.

A decrease in the concentration of total serum lipids occurred near the time of radium treatment in 5 of the 15 patients.

Scattered changes in the concentrations of serum gamma globulin, as measured by the zinc turbidity test, and the occasional development of positive cephalin tests were observed.

Indirect participation of the liver in the systemic response to irradiation is discussed as a possible mechanism of the above phenomena.

Four graphs; 1 table.

Investigation of Radium Deposition in Human Skeleton by Gross and Detailed Autoradiography. William B. Looney and Lois A. Woodruff. Arch. Path. **56:** 1–12, July 1953.

A comprehensive picture of radium deposition in the human skeleton was obtained by the authors by the use of gross and detailed radioautographs. Multiple specimens of fresh human bone from 3 cases provided the source material. One case was that of a forty-eight-year-old white woman who died in 1951 from a fibrosarcoma of the foot. She had been given radium chloride solution ("radium water") for a condition diagnosed as "migratory arthritis" twenty-two years prior to her death. A second patient, a fifty-one-year-old white woman, had a reconstruction operation on the head of the femur for aseptic necrosis in 1951. Seventeen years before the operation she had been given intravenous injections of radium chloride for "mental depression." The third patient was a forty-eight year-old white woman who had worked as a dial painter from 1921 to 1927 and died in 1951. She had multiple femoral fractures, osteomyelitis of the mandible, and other complications which could be attributed to radium poisoning.

The bone specimens obtained were from the femur, humerus, tibia, fibula, skull, vertebra, ilium, and ribs. The gross autoradiographs provided (1) a comprehensive picture of radium distribution throughout the skeleton; (2) the approximate dimensions of individual areas of deposition; (3) the localization of areas for further detailed study.

The gross autoradiographs showed the radium to be irregularly distributed in both compact and cancellous bone in small areas of heavy concentration. Detailed autoradiographs verified the random distribution. Areas of concentration of radium in trabecular bone were 5 to 15 μ in greatest diameter. Occasional concentrations ran parallel to trabeculae for 50 to 100 μ. At the subarticular junction in the long bones and vertebrae, fairly uniform concentrations were found. Some cementing lines were well outlined by significant amounts of radium. In compact bone 10 to 15 per cent of the haversian systems and interstitial lamellae showed areas of radium concentration.

Microscopically, destructive bone changes were found in the absence, as well as in the presence, of radium concentration as seen radioautographically. In the areas of bone destruction without radium, it is believed that the latter had probably been removed, possibly by ion exchange. It is evident that some relation exists between radium deposition and bone destruction, although no definite conclusion could be drawn about a direct relation in view of the long duration of the pathologic process in the cases studied.

Seven autoradiographs; 1 photomicrograph.

RICHARD E. OTTOMAN, M.D.
Los Angeles, Calif.

Effect of Intragastric Irradiation on Gastric Acidity in the Dog. Benum W. Fox, Armand Littman, M. I. Grossman, and A. C. Ivy. Gastroenterology **24:** 517–534, August 1953.

Since roentgen therapy has been found of benefit in the treatment of peptic ulcer in man and has a definite "acid-inhibiting" effect on the gastric mucosa (Ricketts *et al.*: Gastroenterology **11:** 789, 807, 818, 1948. Abst. in Radiology **53:** 776, 777, 779, 1949), intragastric irradiation with radium applicators and beta-emitting isotopes would seem to be desirable and feasible, obviating the necessity for irradiating surrounding tissues and organs.

In an attempt to establish rigid criteria for possible application of these intragastric sources in man, and to

establish a safe technic for the use of isotopes in solid form within the stomach, the authors undertook the experimental studies reported here.

Gastric fistulas were made in 17 dogs, plastic and metal cannulas being introduced, through which the gastric contents could be removed for acid analyses. After the animals recovered from the preparatory surgery, the procedure included: (1) establishment of a series of control histamine tests (0.167 mg. histamine diphosphate per kilo was injected as the stimulus) performed several times a week; (2) irradiation; (3) post-irradiation serial histamine tests.

Three separate radiation sources were used: (1) radium, a gamma source, in 4 Monel needles containing a total of 50 mg., in order that a comparison could be made between beta and gamma effect, (2) ruthenium 106-rhodium 106, and (3) strontium 90, the two latter emitting beta particles of moderately high energy. The isotopes were used in cylinders 2 cm. long and up to 6 mm. in diameter. The sources were fixed in the center of an expandable balloon which, on inflation with 525 c.c. of air, would allow radiation to be delivered to the gastric mucosa at a fairly uniform distance of 5 cm.; 125-millicurie sources were used.

Dosage for the radium applicator was expressed in milligram hours. An extrapolation ionization chamber was designed for measurement of the ruthenium-rhodium surface dose and, at a point perpendicular to its axis at the mid-point, dosage was found to be 991.6 r beta equivalents per hour. Strontium doses were expressed in curie hours.

Three dogs were treated with radium, 6 with $Ru\text{-}Rh^{106}$, 9 with Sr^{90}, and 2 with both $Ru\text{-}Rh^{106}$ and Sr^{90}. The applicators were introduced through the fistulas and via the esophagus by swallowing.

Doses ranging from ineffective to lethal were given with each isotope. For each agent there was found to be a "therapeutic" range of dosage causing a significant reduction of gastric acidity, lasting two to eight months, with minimal side effects. This range appeared to be between 3,000 and 4,500 mg. hr. for radium, about 1,600 rbe (roentgen beta equivalents) for $Ru\text{-}Rh^{106}$ (0.20 curie-hours), and between 0.75 and 1.5 curie hours for Sr^{90}. Achlorhydria to large amounts of histamine occurred only occasionally with radiation doses below the lethal range. Death occurred with 9,000 mg. hr. of radium, 4,760 rbe of $Ru\text{-}Rh^{106}$ and 12 curie hours of Sr^{90}. Because the $Ru\text{-}Rh^{106}$ was plated on a metal cylinder, it was the safest to handle and thus warrants further experimental study. Results are presented graphically and in tabular form.

Seventeen graphs; 1 diagram; 4 tables.

JOHN F. RIESSER, M.D.
Springfield, Ohio

Radon Inhalation Studies in Rats. S. H. Cohn and J. K. Gong. Arch. Indust. Hyg. **7:** 508–515, June 1953.

With increased and intensified uranium-mining operations, the evaluation of the radiation hazard to personnel from radon inhalation has become of greater significance. The authors conducted experiments on rats with a view to comparing the radiation delivered to the respiratory system after the inhalation of radon plus its degradation products and the radiation delivered after the inhalation of the same atmosphere containing radon but with the decay products filtered. An effort was also made to determine the differential distribution of activity in various parts of the respiratory tract following inhalation of radon.

The results of the study are summarized as follows: "In calculating the relative amount of radiation to the lungs of animals exposed to a radon atmosphere, the decay products of radon which would also be present in this atmosphere must be considered of primary importance. The radiation flux to the respiratory tract from inhaled radon is considerably less than that from the inhaled decay products, RaA, RaB, RaC, and RaC'. This is due to the filtering out by the lungs of those dust particles which contain the radon decay products and the accumulation of these radioactive substances in the lung.

"There is an unequal distribution of the radon decay products within the lung, apparently as a function of the size of the dust particles upon which the daughters are deposited. After inhalation of a radon-daughter atmosphere, only a small amount of the total gamma activity in the respiratory system was found in the trachea and large bronchi. However, the radiation exposure per unit surface area was much greater for the bronchi than for other lung tissue.

"It is obvious that by instituting measures for the constant removal of dust in mines or by having workers wear masks that prevent the inhalation of normally present atmospheric dust, the hazard from inhalation of radon gas can be greatly reduced. It would appear that inhalation studies which consider the radiation from radon gas alone do not provide a realistic approach to the problem insofar as hazards to humans are concerned."

Two photographs; 4 graphs.

Protecting Action of Citrate on the Agent of Chicken Tumor I (Rous Sarcoma Virus) During Roentgen Radiation in Vitro. W. Ray Bryan, Egon Lorenz, and Dorothy Calnan. J. Nat. Cancer Inst. **13:** 1447–1461 June 1953.

The authors' own conclusions and summary cannot be improved upon and are qnoted:

"Suspensions of the agent of chicken tumor I (Rous sarcoma virus) in citrate solutions of varying molar concentration were irradiated with total doses of 0.7×10^5 r or 1.4×10^5 r of X radiation, and determinations of residual biological activity were made at 0, 4, and 24-hour intervals following irradiation. Similar determinations were made, also, on irradiated suspensions of the agent in distilled water and in citrate solution containing 2 per cent normal horse serum. Additional control observations involved the treatment of active preparations of the tumor agent with previously irradiated suspending solutions, the same as above.

"It was found that citrate in 1.6×10^{-2} M or higher concentration in the suspending solution protected the agent to a large extent against immediate inactivation by indirect effects of the X radiation, but that a slow or delayed reaction followed which resulted in a progressive fall in residual biological activity over a period of at least 24 hours. The immediate protection by citrate, in maximally effective concentrations, was systematically less than that provided by normal horse serum. Successive decreases in citrate concentration below 1.6×10^{-2} M gave progressively less immediate protection against the indirect irradiation effects, down to the lowest concentration tested, namely $10^{-0.5}$ M.

"The findings are discussed and certain of the data

involving direct irradiation effects are compared with similar data previously obtained."

Nine charts. GEORGE A. SHIPMAN, M.D.
New Orleans, La.

Occurrence of Pulmonary Tumors in Strain A Mice Following Total-Body X-Radiation and Injection of Nitrogen Mustard. W. E. Heston, Egon Lorenz, and Margaret K. Deringer. Cancer Res. **13**: 573–577, August 1953.

An investigation was undertaken to ascertain whether or not an additive effect of x-irradiation and nitrogen mustard could be demonstrated in the induction of pulmonary tumors in strain A mice. The incidence of pulmonary tumors in untreated mice of this strain is approximately 90 per cent in animals eighteen months of age and 50 per cent in animals twelve months of age. Two hundred fifty-six mice two and three months of age were distributed in four groups: Group I, the controls; Group II, those to receive irradiation; Group III, those to receive nitrogen mustard; Group IV, those to receive irradiation plus nitrogen mustard. The mice of Groups II and IV were given a single total-body exposure of 900 r, with the following factors: 186 kv.p., 20 ma., 0.25 mm. Cu plus 1.06 mm. Al filtration. Two tubes opposite each other were used to obtain a uniform tissue dose; their foci were 54 cm. from the center of the mice. This gave a dosage rate of 93 r per minute. Since the dose of 900 r is 100 per cent lethal, the spleens of the mice were surgically exteriorized and shielded in lead chambers. The mice in Groups I and III were subjected to a mock spleen-shielding operation. Ten days later, the animals in Group III and those of Group IV were each given an injection of 0.025 mg. nitrogen mustard in 0.25 c.c. distilled water. Two days later these animals were given a second similar injection.

The occurrence of pulmonary tumors in the mice exposed to 900 r with spleens shielded was found to be comparable to that in control animals that had been subjected only to a mock spleen-shielding operation.

There was a significant increase in the occurrence of pulmonary tumors in strain A mice that had been injected with 0.05 mg. nitrogen mustard following a mock spleen-shielding operation.

In strain A mice that had received 900 r with their spleens shielded and subsequently been injected with 0.05 mg. nitrogen mustard, the occurrence of pulmonary tumors was not significantly different from that of the controls or that of the group receiving irradiation alone, and was significantly less than that of the group receiving nitrogen mustard alone.

One chart; 1 table.

Secondary Evolution of Molecules Having a Phenol Radical, Following Roentgen Irradiation. J. Loiseleur and M. Sauvage. Compt. rend. acad. sc. **237**: 204–206, July 1953. (In French)

Action of Reducing Substances on the Latent Radiation Effect in Vitro. J. Loiseleur, L. Catinot, and P. Morenne. Ibid., pp. 410–412, August 1953. (In French)

Chemical Synthesis Following the Action of Roentgen Rays. A. Lacassagne and J. Loiseleur. Ibid., pp. 417–419, August 1953. (In French)

Chemical Synthesis Following the Action of Physical Peroxidase Agents (Roentgen Rays, Ultraviolet and Ultrasonic Rays). Ibid., pp. 461–462, August 1953. (In French)

These four short communications to the French Academy of Sciences emanate from the Laboratories of Radiobiology of the Radium Institute of the University of Paris; they construct a brilliant follow-up to previous experiments from the same source on the role of oxygen in radiobiology.

In aqueous solution molecules presenting a phenol radical show a considerable increase of their optic density following irradiation in the presence of oxygen. The effect continues in the hours that follow irradiation and is proportional to the x-ray dose. The presence of copper in very light solutions results in a catalytic effect. Heat accelerates the "latent" effects.

The "latent" effects of irradiation of these phenol molecules is diminished or suppressed by the introduction of a reductor substance in the solution following irradiation. The reductor substance has a lesser or greater counter effect, depending on pH.

The irradiation results in opposite effects depending on the molecular mass: the complex molecules of high molecular weight undergo *desmolysis*, whereas the simple molecules of low molecular weight undergo condensation and *synthesis*. The question is raised as to whether or not this synthesis plays a role in the carcinogenic effects of radiations. At any rate, it appears demonstrated that radiations can produce the complicated synthesis of organic compounds: methylene blue as well as other substances have been visibly synthesized *in vitro*.

The synthesizing effects of irradiation are also produced by other physical agents, such as ultraviolet waves and ultra sound waves, although the effects vary in degree. Two conditions appear necessary for a synthesizing effect: the presence of oxygen and of a molecule of low molecular weight. The only difference in the effects with the various physical agents is the ratio of the energy spent to the intensity of the reaction produced; the ratio is most favorable to the effects of ultraviolet rays. J. A. DEL REGATO, M.D.
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Field Equipment for the Collection and Evaluation of Toxic and Radioactive Contaminants. W. B. Harris, H. D. LeVine, and M. Eisenbud. Arch. Indust. Hyg. **7**: 490–502, June 1953.

This report from the Health and Safety Division of the U. S. Atomic Energy Commission describes equipment for the collection and evaluation of toxic and radioactive contaminants in air. Mechanical filtration was chosen as the most satisfactory means of sampling air containing microgram quantities of dust, and dry impingement for determining particle size. Among the instruments described are a beta filter paper meter and a radon integrating chamber and analyzer.

Fourteen illustrations.